A PROSPECTIVE STUDY OF CLINICAL PROFILE OF EMPHYSEMATOUS PYELONEPHRITIS IN TYPE 2 DIABETES MELLITUS

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KILPAUK MEDICAL COLLEGE HOSPITAL

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CERTIFICATE

This is to certify that the dissertation entitled "**A PROSPECTIVE STUDY OF CLINICAL PROFILE OF EMPHYSEMATOUS PYELONEPHRITIS IN TYPE 2 DIABETES MELLITUS"** is the bonafide work of Dr.V.Nandakumar in partial fulfillment of the university regulations of Tamil Nadu Dr. M.G.R. University, Chennai, for MD (Branch I) General Medicine examination to be held in April 2013**.**

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DECLARATION

I, **Dr. V.Nandakumar,** hereby declare that, I carried out this work entitled "**A PROSPECTIVE STUDY OF CLINICAL PROFILE OF EMPHYSEMATOUS PYELONEPHRITIS IN TYPE 2 DIABETES MELLITUS"** at Govt. Kilpauk Medical College Hospital, under the guidance of **Prof. Dr. T. Ravindran, MD,** Professor of Medicine. I also declare that this bonafide work has not been submitted in part or full by me or any others for any award, degree or diploma to any other University or Board either in India or abroad.

This is submitted to The Tamil Nadu Dr. M.G.R. University, Chennai, in partial fulfilment of the university rules and regulations of for MD degree examination in General Medicine (Branch I) to be held in April 2013.

Place: Chennai

Date: **Dr. V.Nandakumar**

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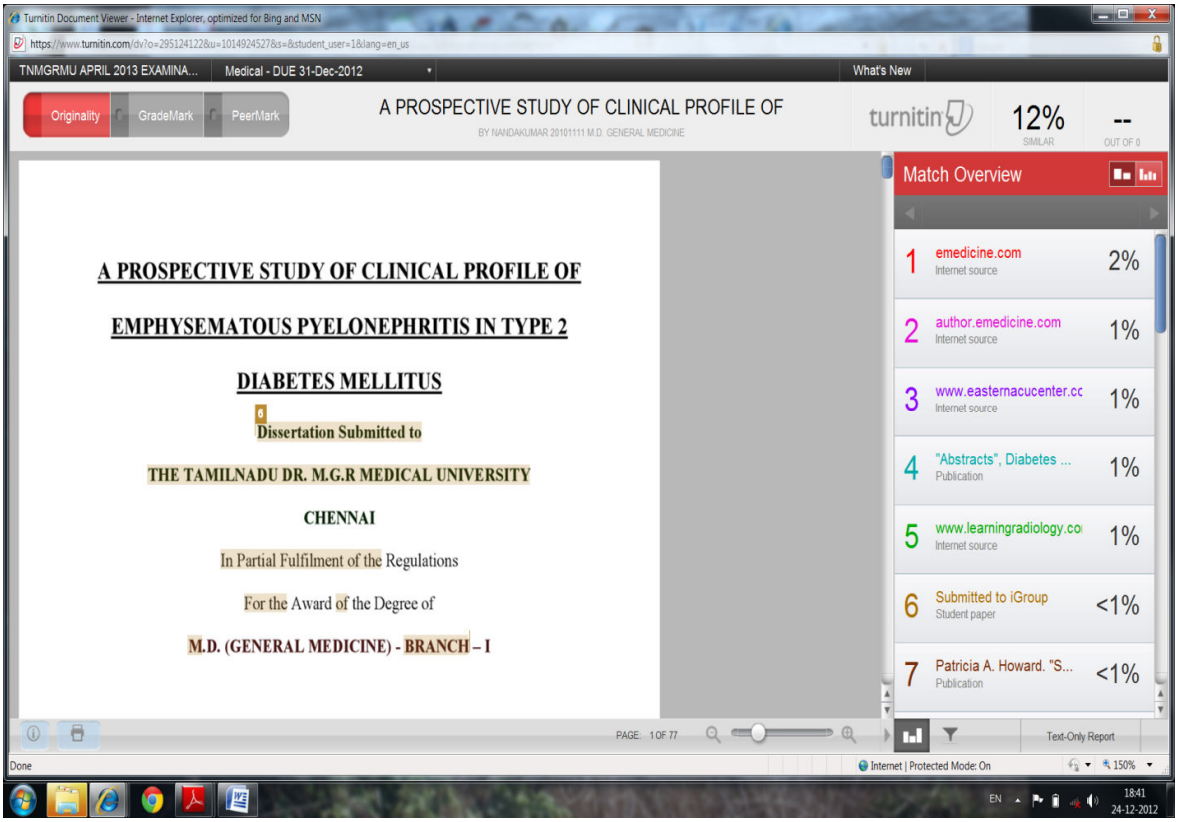
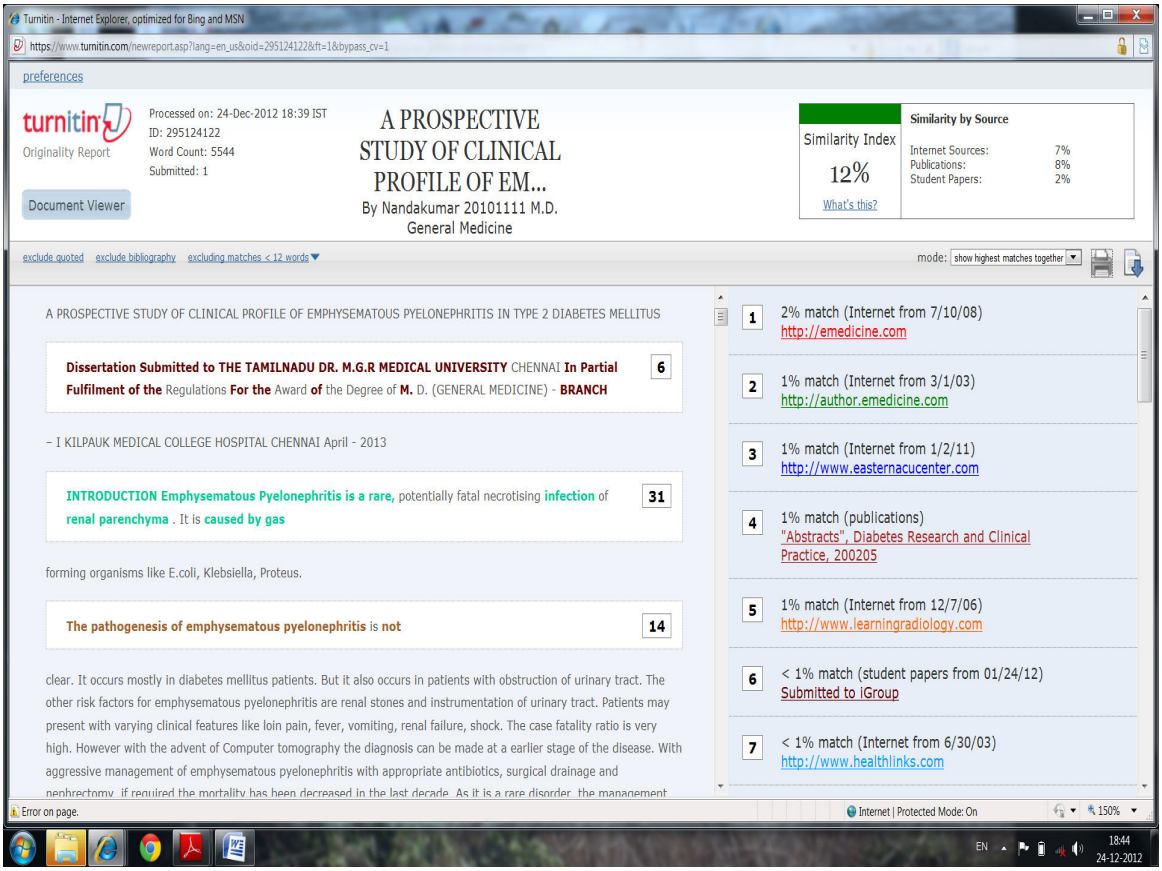
Last, but not the least, my profound gratitude to all the patients, to whom I owe everything because, this venture would not have been possible without them.

TURNITIN ANTI- PLAGIARISM SITE SCREEN SHOT



TURNITIN ANTI- PLAGIARISM SITE

ORIGINALITY REPORT



ETHICAL COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE, KILPAUK,
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Venue: PANAGAL HALL, KMC
Dt: 01.02.2011

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Sub: Ethical Committee project work - approved – regarding.
Ref: Lr.No.3944/Audit/E1/09 Dt. 30.11.2010

****

With above reference, the Institutional Ethical committee meeting for the following students was conducted at our Institution on 01.02.2011.

S.NO.	Name	Topic
1.	Dr.Navin Kumar, MS(Ortho), PG., Govt. Royapettah Hospital, Chennai.	1.To Identify a Safe Zone to approach proximal Humerus 2.To study Anatomical relations of Axillary nerve, its course & its Variations
2.	Dr.T.Satheesh Kumar, D.Ortho., PG., Govt. Royapettah Hospital, Chennai	Heriditary Multiple Exostosis
3.	Dr.J. Jeya Shambavi, MD(Pathology), PG., Govt. Kilpauk Medical College, Chennai-10	Clinicopathological Histomorphological and Immunohistochemical Study of Neuroendocrine Tumors of GIT
4.	Dr.L. R. Saranya. MD., (Paed.)PG., Govt. Kilpauk Medical College, Chennai-10	Cord Blood Zinc Level in Term-Small for Gestational Age Neonates
5.	Dr.A.Satheesh Kumar, MS(ENT), PG., Kilpauk Medical College, Chennai	Study on Cases of Chronic Suppurative Otitis Media in Tubo Tympanic Type Due to Sinusitis as Focal Sepsis
6.	R.Prathiban,(Msc.,Physiology), PG., Student, The TN. Dr.MGR Medical University,Chennai-32	Prevalence of Cardiac Dysautonomia in Type I Diabetes mellitus
7.	B. Manikandan, (Msc., Physiology), PG., Student, The TN Dr.M.G.R. Medical University, Chennai-32.	A Comparative Study of Left Ventricular Structure and Function in Obese and Non Obese Subjects
8.	G. Selvakumar, (MSc., Physiology), PG., Student, The TN Dr.M.G.R. Medical University, Chennai-32.	A Study of the Intraocular Pressure In Patients with Diabetic Normotensive, Diabetic Hypertensive and Normal Subjects

9.	R. Ragulji, (Msc.,Physiology), PG., The TN Dr.MGR Medical University, Chennai-32.	A Study of Pulmonary function in insulin dependent diabetes mellitus
10.	V.M. Jenila Veny,(MscPhysiology), PG. The TN Dr.MGR Medical University, Chennai-32	Cardiovascular Autonomic Dysfunction in Chronic Kidney Disease
11.	Dr.G. Lakshmi, MD(O&G), PG., Govt. Kilpauk Medical College,Chennai-10	A Study of Association of Thyroid Disorders in Abnormal Uterine Bleeding
12.	Dr.R. Harini, MD(O&G), PG., Kilpauk Medical College, Chennai	Single Dose Antibacterial treatment for Asymptomatic Bacteriuria in Pregnancy
13.	Dr.E.Geetha, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	A Study of the incidence course of Pregnancy and Pregnancy outcome in Obstetric Cholestasis and to evaluate the efficiency of UDCA in relieving the Symptoms and Improving the Perinatal outcome in these Patients
14.	Dr.S. Nithya, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	Prospective Study of Prevalence of diabetes Mellitus, Thyroid Dysfunction and Hyperprolactinemia in Recurrent Pregnancy loss
15.	Dr.Mohideen Fathima, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	A Study of evaluation of multi system changes in Gestational hypertension / severe pre-eclamptic/eclampasia patients
16.	Dr.M.Padma Priya, MD(O&G), PG., Kilpauk Medical College, Chennai	Dyslipidemia as a Predictor of PIH
17.	Mrs.G. Savitha, (Msc.,Medical Bio Chemistry), TN Dr.M.G.R.Medical University, Chennai-32.	Association of subclinical hypothyroidism in metabolic syndrome patients
18.	Dr.K. Bharadhwaj, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study on Peripheral Vascular Disease in Type 2 Diabetes Mellitus
19.	Dr.B.Priya, MD(G.M.), PG	Study of Serum Bilirubin Concentration in Established Coronary Artery Disease
20.	Dr.R.Hema, MD(G.M.), PG.,	Study of Troponin I level in Supraventricular Tachycardia in Non Cad Patients
21.	Dr.P.Manoj Kumar, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study on Pulmonary Functions in Type 2 Diabetes Mellitus
22.	Dr.M.Dhanasekar, MD(G.M.), PG.,	Prognostic Risk Stratification of Acute Coronary Syndrome – Role of Highly Sencitive – Reactive Protien
23.	Dr.N. Karthik, MD(G.M.), PG., Govt.Kilpauk Medical College, Chennai-10	A Study of Comparison of QT Dispersion in Acute Myocardial Infraction Between Early Reperfusion and Late Reperfusion Therapy

24.	Dr.H. Anuradha, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study of Stress Hyperglycemia in Moderate Degree Burns
25	Dr. V. Nandakumar, MD(G.M.), PG.,	A Prospective Study of Clinical Profile of Emphysematous Pyelonephritis in Type Two Diabetes Mellitus
26.	Dr.S.Sasikumar, MS(G.S.), PG., Govt. Royapettah Hospital, Chennai	A Study of Unusual Presentations of Appendicitis.
27.	Dr.S.R.Padmanabhan, MS(GS), PG., Govt. Royapettah Hospital, Chennai	A Comparative Study Between Autologous Platelet Rich Plasma and Saline Dressing for Diabetic Ulcer
28.	Dr.C.Rose, Scientist-G and Head, Biotechnology, Central Leather Institute, Chennai.	Wound healing efficacy of the chitosan – containing collagenous biomaterial, on burn wound
29.	E.K. Lavanya,B.Tech, Biotechnology, PG., Prathyusha Institute of Technology and Management, Tiruvallur.	Isolation and Characterization of Bacterial Pathogens from Eye Infection

We are glad to inform you that at the Ethical Committee meeting, the documents were discussed and the above short term projects are Ethically approved.

4/2/11

CHAIRPERSON
. DEAN
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Chennai-10.

To: The Individuals

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INTRODUCTION

Emphysematous Pyelonephritis is a rare, potentially fatal necrotising infection of renal parenchyma . It is caused by gas forming organisms like E.coli, Klebsiella, Proteus. The pathogenesis of emphysematous pyelonephritis is not clear. It occurs mostly in diabetes mellitus patients. But it also occurs in patients with obstruction of urinary tract. The other risk factors for emphysematous pyelonephritis are renal stones and instrumentation of urinary tract.

Patients may present with varying clinical features like loin pain, fever, vomiting, renal failure, shock. The case fatality ratio is very high. However with the advent of Computer tomography the diagnosis can be made at a earlier stage of the disease. With aggressive management of emphysematous pyelonephritis with appropriate antibiotics, surgical drainage and nephrectomy, if required the mortality has been decreased in the last decade.

As it is a rare disorder, the management protocols and prognosis are rarely been studied. In our study, we evaluate the predisposing factors clinical presentation, causative organism, mode of therapy of emphysematous pyelonephritis in type 2 diabetes mellitus patients.

AIM OF THE STUDY

1. The aim of this study is to evaluate the

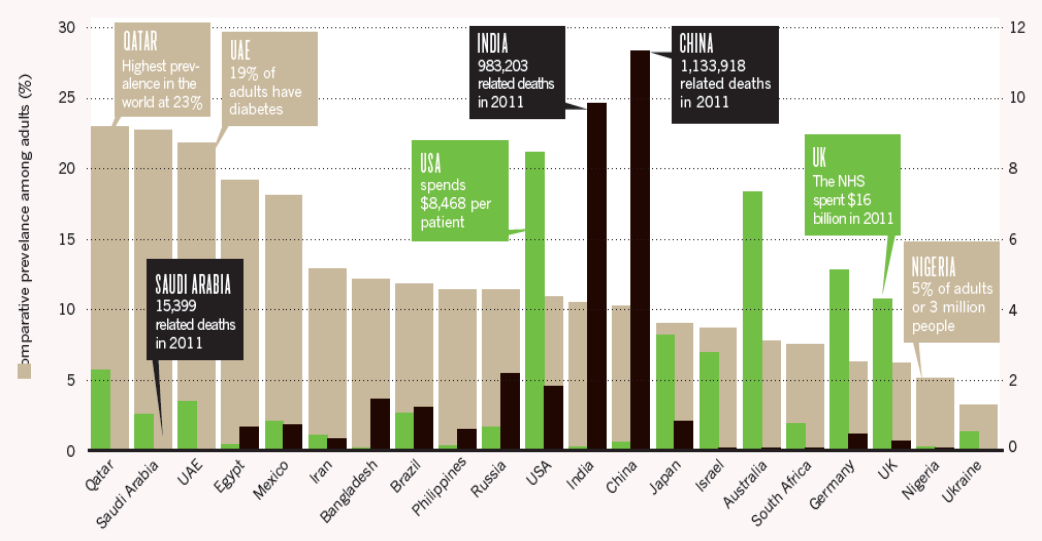
- Predisposing factors
- Clinical features
- Causative organism
- Mode of therapy required for emphysematous pyelonephritis in type 2 diabetes mellitus patients.

2. To evaluate the outcome of emphysematous pyelonephritis in type 2 Diabetes mellitus in our population.

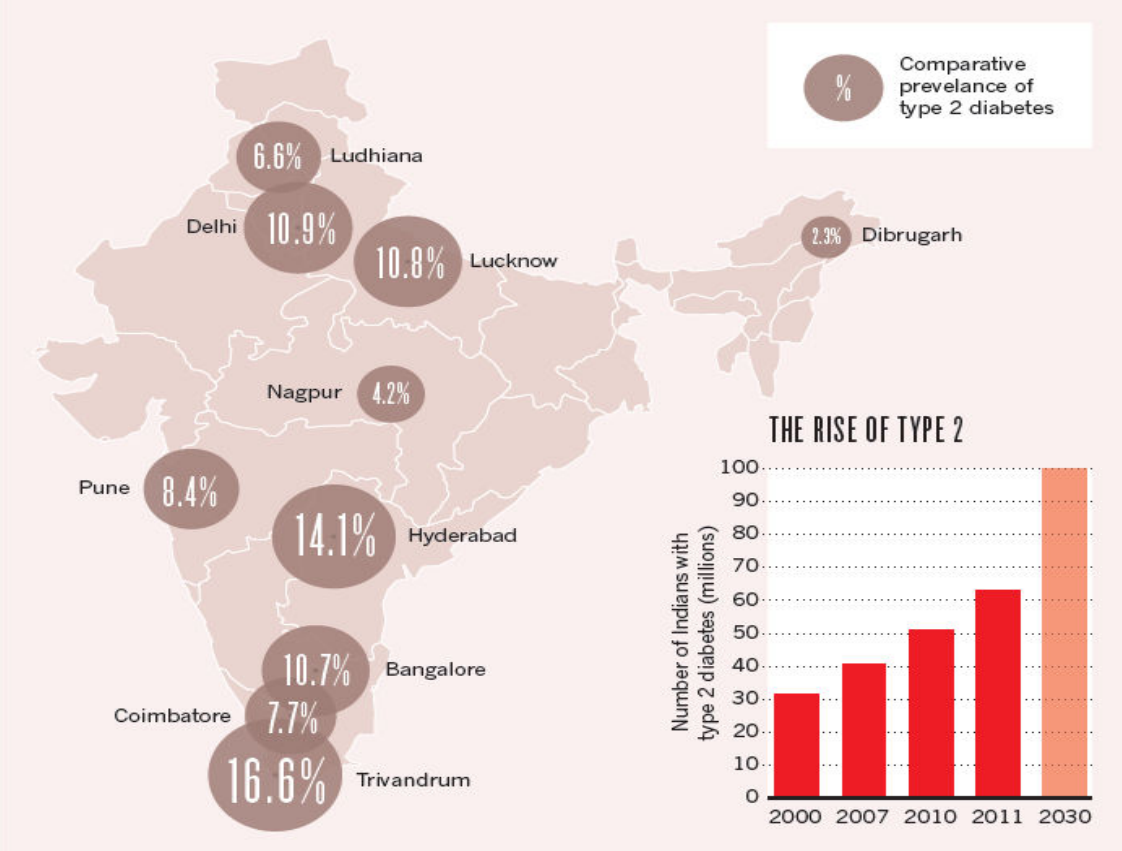
REVIEW OF LITERATURE

Diabetes mellitus is probably the most widely prevalent metabolic disorder among the world population. The incidence of diabetes mellitus is increasing day by day both in global and Indian scenario. The prevalence of diabetes is 2.8% in 2000, but this figure will reach 4.4% in 2030 worldwide.

WORLD WIDE SCENARIO OF DIABETES



Prevalence in Indian scenario: Increasing trend



RISING TREND OF T2DM IN URBANIZED INDIAN CITIES AND THEIR PROJECTED OUTLOOK [I]

With India embracing both eastern and western lifestyle the prevalence of obesity has increased alarmingly, prevalence is between 10 and 50% in various Indian cities. One study from urban Chennai has showed a prevalence rate of 22.8% in males and 31.8% in females[74].

As of 2011 there are 61million diabetics in India. Recent Chennai Rural Urban Epidemiological Study [CURES] had showed a crude prevalence rate of 14.3% [age adjusted],with in a span of 14 years the prevalence had increased alarmingly by 73%[75].

DIAGNOSTIC CRITERIA FOR DIABETES MELLITUS

TABLE 1 : DIAGNOSTIC CRITERIA FOR DIABETES MELLITUS

- Symptoms of diabetes + RBS ≥ 11.1 mmol/dl (200 mg/dl)[a] OR
- FBS ≥ 7.0 mmol/dl (126 mg/dl)[b] OR
- HbA1c > 6.5 % OR
- Two- hour plasma glucose ≥ 11.1 mmol/dl (200 mg/dl)[c] during OGTT.

[a]RBS(Random blood sugar) is without regard to time since last meal.
[b]FBS(Fasting Blood Sugar) after no caloric intake for at least 8 hours.
[c]Blood sugar after 2 hours of glucose load containing 75gms of anhydrous glucose in water

Source : American Diabetes Association, 2011

VALUE	NORMAL	HYPERGLYCEMIA	
		PRE-DIABETES	DIABETES MELLITUS
		Impaired fasting glucose/ impaired glucose tolerance	
FPG	<100 mg/dl	100 – 125 mg/dl	≥126 mg/dl
2h PG	<140 mg/dl	140 – 199 mg/dl	≥200 mg/dl
HbA1c	<5.6 %	5.7 – 6.4 %	> 6.5 %

- ***GLYCOSYLATED HEMOGLOBIN (HbA_1C)***

Glycation refers to non-enzymatic addition of a sugar residue to an amino group of a protein. Hemoglobin, plasma proteins, lens protiens etc., may undergo glycolysation. HbA_1C forms the major fraction (80%). In HbA_1C the N-terminal valine residue of each beta chain gets glycated.

HbA_1C gives a retrospective index of integrated plasma glucose values over a six to eight weeks period. HbA_1C serves as a reliable indicator of diabetic control during the past 90 days, effectiveness of treatment and risk of development of acute or chronic complications. Hence HbA_1C should be performed in all patients with

diabetes during the initial visit and every three months to assess metobolic control .

Normal HbA1C values and interpretations

- Normal range 4.5 – 5.8%
- Risk of hypoglycemia < 4.5%
- Diabetic range > 6.5%
- Prediabetic range 5.8 – 6.5%

HbA1C %	**Mean plasma glucose (mg/dl)**
5	97
6	126
7	154
8	183
9	212
10	240
11	269
12	298

DIABETES AND KIDNEY

Diabetes has become the most common cause for chronic kidney disease all over the world. In type 1diabetes, nephropathy occurs 25 to 40 years after diagnosis and his seen in about 25 to 40% of patients. In type 2 diabetes, nephropathy will be present in 5 to 10% of patients at the time of diagnosis itself. The cumulative incidence is 25% after 20 years of diagnosis.

Diabetic nephropathy is a clinical syndrome characterized by persistent albuminuria (> 300 mg / 24 hours or > 200 mcg / min) on atleast 2 occasions separated by 3 – 6 months. Albuminuria > 300 mg / 24 hours is equivalent to a total proteinuria > 500 mg / 24 hours. This is also called as **macroalbuminuria.** Urine albumin excretion of 30 – 300 mg / 24 hours or 20 – 200 mcg/ min is **microalbuminuria.**

Stages of diabetic nephropathy

Carl Eric Mogensen's classical description in 1983 of the five stages of diabetic nephropathy was for type 1 diabetes mellitus. However the general pattern remains the same for type 2 diabetes mellitus. The stages are as follows:

Stage 1 – Stage of renal hypertrophy and glomerular hyperfiltration

Stage 2 – Stage of apparent normalcy

Stage 3 – Stage of microalbuminuria or incipient nephropathy

Stage 4 – Stage of established nephropathy

Stage 5 – End stage renal disease

Pathogenesis of diabetic nephropathy

The primary insult to the kidneys is due to hyperglycemia. Hypertension and proteinuria worsens the nephropathy. It is postulated that increase in glucose leads to oxygen free radicals generation that result in nephropathy. Four pathways - protein C kinase pathway, polyol pathway, hexoaminase pathway and formation of advanced glycation end products result in over production of cytokines which induce fibrosis and increase vascular permeability. Accumulation of protein in tubular cells initiates mesenchymal cell transformation leading to chronic tubular injury.

Three main changes occur in diabetic nephropathy. First is thickening of glomerular basement membrane. Second is glomerular sclerosis. Third is appearance of Kimmelstiel-wilson nodules.

INFECTIONS ASSOCIATED WITH DIABETES MELLITUS

Infections associated with diabetes mellitus can be discussed under two headings.

- **Infections with an increased prevalence in patients with DM**
  - Oral and esophageal candidiasis
  - Bateriuria and cystitis
  - Pyelonephritis and perinephric abscess
  - Surgical site infection
  - Cellulitis and osteomyelitis
  - Pyomyositis
  - Tuberculosis of lung
  - Staphylococcal and gram negative pneumonia

- **Infections unique to patients with DM**
  - Rhinocerebral mucormycosis
  - Malignant otitisexterna
  - Synergistic necrotizing cellulitis
  - Fournier's gangrene
  - **Emphysematous cystitis**
  - **Emphysematous pyelitis**
  - **Emphysematous pyelonephritis**

EMPHYSEMATOUS PYELONEPHRITIS

Emphysematous pyelitis is the term used to describe the presence of gas limited through renal excretory system. Emphysematous cystitis is due to inflammation of bladder mucosa and its musculature. Emphysematous pyelonephritis is defined as necrotizing infection of renal parenchyma and its surrounding tissues. Gas accumulates in renal parenchyma, collecting system or perinephric tissues. It carries a mortality of 100% if left untreated.

History and epidemiology emphysematous pyelonephritis

Emphysematous pyelonephritis was first reported in 1898 by Kelly & Mac Cullum as a case of pneumaturia as they found it is a gas forming renal infection. Since then various names like renal emphysema, pneumonephritis, are used. In 1962 Schultz & Kloferin proposed the name emphysematous Pyelonephritis.

Epidemiology

The average age of presentation of patients with emphysematous pyelonephritis is 56 years, although the range may vary from 16 - 80 years. Females are affected more commonly than males. The reason for female preponderance may be due to increased risk of genitourinary infections

in them. The male : female ratio is 7:1. Right kidney is less commonly affected than the left may be due to preponderance of left sided urinary tract obstruction. In ten percent of cases both the kidneys are involved.

Almost all the patients are diabetic with poor blood sugar control with elevated glycosylated hemoglobin. Rarely few cases occurs in non – diabetic population . Other risk factors for this rare disease are renal failure, obstructed urinary tract, previous history of instrumentation, renal stones, polycystic kidneys. The main cause of emphysematous Pyelonephritis in patients without diabetes is obstruction of urinary tract.

Etiology and pathogenesis

It is caused by gas forming organisms . E. coli is the most common organism.

Common organisms include:

- Escherichia coli (68%)
- Klebsiella pneumonia (24%)
- Proteus mirabilis
- Pseudomonas

Rare organisms include:

- Clostridium perfrigens
- Citrobacter
- Aerobacter aerogenes
- Candidiasis

The exact mechanism of gas formation is not clear. The proposed cause for gas chamber formation are

- Increased gas production
- Due to poor blood supply there is decreased transport of gas
- Formation of gas chamber
- Equilibrium of gas chamber and tissue gas
- Expansion or collapse of gas chamber

Regarding the gas content many hypothesis are proposed. Usually bacteria get their energy by fermentation of glucose through glycolytic pathway. In 1889 nitrogen, hydrogen, and Carbondioxide were found in a patient with pneumaturia. The gases demonstrated are

- Nitrogen - 60%
- Hydrogen – 15%
- Carbondioxide - 5%

- Oxygen – 5%
- Rarely methane, ammonia are also demonstrated in some studies.

Gas forming organism follow two different pathways namely,

1. Mixed fermentation - E. coli, Klebsiella, Proteus – Carbon dioxide
2. Butyric fermentation – Clostridium - hydrogen

Fig - Macroscopic appearance showing diffuse parenchymal necrosis

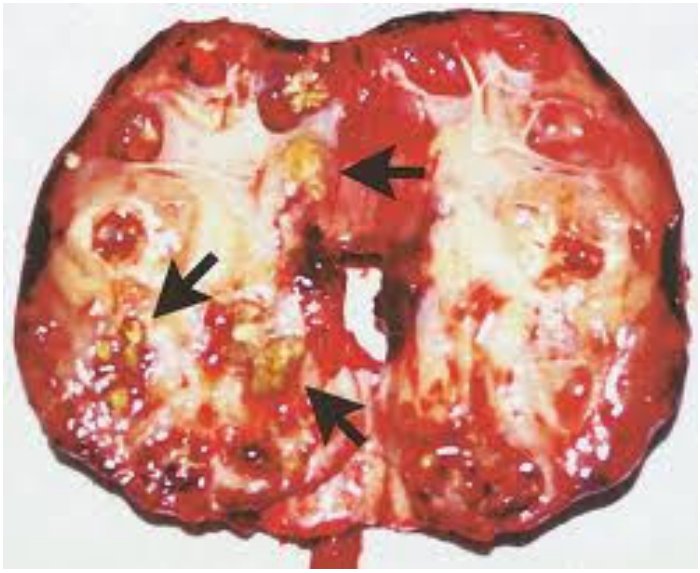


Fig - Cut cross section shows diffuse parenchymal necrosis and haemorhage

The pathogenesis of emphysematous pyelonephritis is poorly understood . Renal infarcts, thrombosis, abcess and necrosis are demonstrated in pathological specimen of emphysematous pyelonephritis. Four reasons are described for the occurrence of emphysematous pyelonephritis are

- Bacteria that forms gas
- Increased bacterial growth due to high blood sugar
- Decreased tissue perfusion that leads to tissue ischemia and necrosis
- Defective immune response

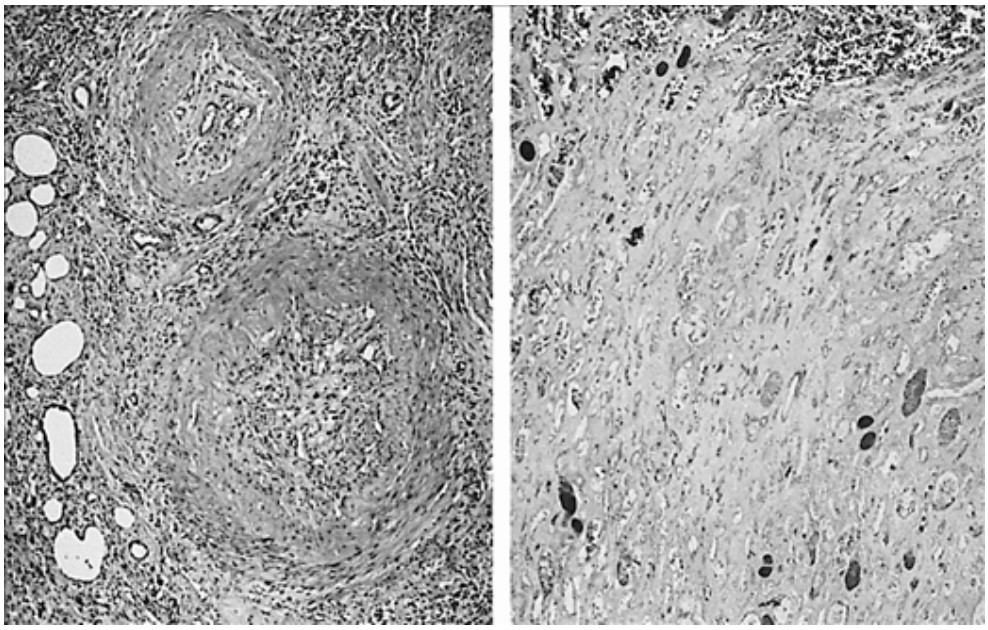


Fig - Pathological specimen showing areas of renal infarction, vascular thrombosis in a patient with emphysematous pyelonephritis.

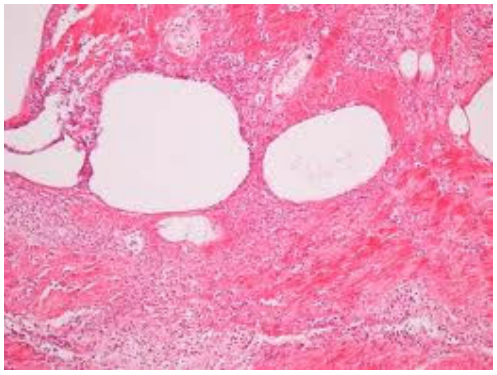


Fig - Histology showing renal infarction and glomerularscelorsis

E. coli and emphysematous Pyelonephritis

E. coli is the most common organism causing both complicated (21 – 54%) and uncomplicated (70 – 95%) urinary tract infection. It

accounts for 68% of cases of emphysematous Pyelonephritis. The various mechanisms by which E.coli causes emphysematous pyelonephritis are

- O serotypes interfere with compliment dependent bacterial killing.
- K antigens (capsular polysaccharide) or anti phagocytic and anti complimentary
- P fimbriae are mannose resistant. The receptor binding adhesin at the tip of P fimbriae is pab G and there are three classes of G – tip proteins. Class II – tip adhesin is associated with emphysematous Pyelonephritis. Class III– tip adhesin is associated with cystitis.

Other virulence factors of E.coli identified by Polymerase chain reaction:

- iroN, iron – regulated gene.
- Iha, homologue adhesion
- kpsMT, group II capsule
- ompT, outer membrane protease T
- USP , uropathogenic protein

Clinical features

Most of the patients with emphysematous pyelonephritis presents with pyrexia, pain over flank and vomiting. These clinical features do not differentiate upper urinary tract infection from emphysematous Pyelonephritis. Crepitus over the flank area may occur in late stages, but is characteristic.

The initial presentations of late stages of emphysematous Pyelonephritis may be

- Hypotension
- Decreased platelet count
- Altered sensorium
- Worsening renal function
- Subcutaneous emphysema
- Pneumomediastinum

Associated comorbid conditions include alcoholism, diabetic ketosis, renal stones. Emphysematous pyelonephritis may complicate pregnancy. It does not affect pediatric diabetic population. High index of suspicion is required for early diagnosis of this potentially fatal condition because late stages of this disease is associated with higher mortality.

The clinical features, routine blood and urine investigations are mentioned in the following table.

Table 1. Clinical Features and Laboratory Data at Initial Presentation

Variable	No. (%) of Patients
Clinical features	
Fever	38 (79)
Flank, abdomen, or back pain	34 (71)
Nausea, vomiting	8 (17)
Dyspnea	6 (13)
Acute renal function impairment	17 (35)
Disturbance of consciousness*	9 (19)
Shock	14 (29)
Laboratory data	
Glycosylated hemoglobin A_{1C} >0.08†	21 (72)
Leukocytosis (leukocyte count $>12 \times 10^9$/L)	32 (67)
Thrombocytopenia (platelet count $<120 \times 10^9$/L)	22 (46)
Urinalysis	
Pyuria	38 (79)
Macrohematuria (RBCs >100 per HPF‡)	6 (13)
Severe proteinuria§ (>3 g/L)	10 (21)

If the patient presents with alteration of sensorium , hypotension, elevated renal parameters, low platelet count the prognosis is found to be poor. Patients presenting with poor blood sugar control with diabetic ketosis also carry a poor prognosis. If patients have adequately controlled blood sugar level and hence HbA_1C carries good prognosis.

Laboratory investigations

Complete blood count will show increased total count with neutrophilia with a left shift. Platelet counts are decreased. Thrombocytopenia is one of the poor prognostic factors. Peripheral smear study will suggest features of sepsis.

Routine urine examinations will reveal plenty of pus cells, albuminuria which may be either due to acute kidney injury or preexcisting diabetic nephropathy. Urine spot protein creatinine ratio will be increased. Urine acetone positivity will suggest poor prognosis.

Urine culture will reveal the causative organism. Urine culture is positive in most of the patients. Antibiotics should be given according to sensitivity pattern. E.Coli, Klebsiella, Proteus are the common organisms grown in urine culture.

Blood sugar is elevated in most of the cases. HbA_1C value will reveal the past three months sugar control. Usually HbA_1C is elevated. Increased creatinine may suggest preexisiting diabetic nephropathy or acute renal injury. Again increasing creatinine values carry grave prognosis.

Blood culture is positive in forty to fifty percent of patients. Positive blood culture carries poor prognosis. Antibiotics should be given according to sensitivity pattern. Mixed culture pattern rarely occurs.

Imaging studies

Patients should be stabilised with fluids and appropriate antibiotics before radiological investigations. Emphysematous Pyelonephritis is classified radiologically using plain X ray KUB, Ultrasonogram KUB, non contrast CT KUB .

Plain X ray KUB

Langston and Pfsiter described three main radiological patterns

- Diffuse mottling of the renal parenchyma
- Bubbly renal parenchyma surrounded by crescent shape gas in perinephric space
- Extension of gas through gerota fascia

Michaeli suggested three stages

- Stage I – Gas within renal parenchyma or in the perinephric tissue
- Stage II – Gas is present in the kidney and its surroundings

- Stage III – Extension of gas through gerota fascia or bilateral disease

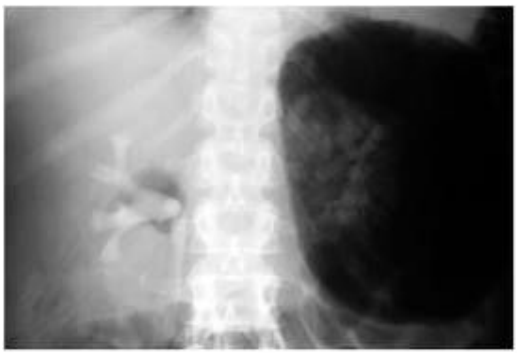


Fig - Stage III disease involving left kidney

Fig - Right kidney appears normal. Bowel gas shadow seen

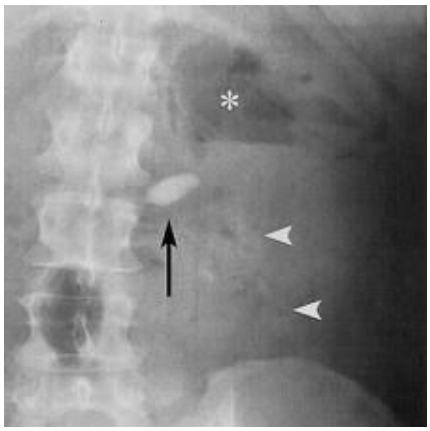


Fig – Dark arrow shows 2 cm calcification in left ureteropelvic junction White arrows show small calcifications. shows large air fluid level in left upperpole .

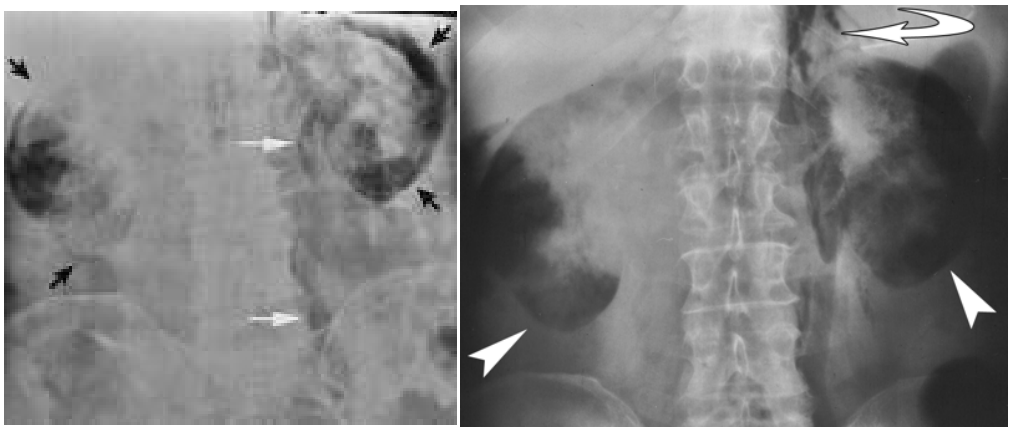


Fig – X ray shows bilateral Emphysematous pyelonephritis Stage III disesase

Ultrasonogram KUB

- Ultrasound findings.
    - High-amplitude echoes within renal sinus and/or renal parenchyma associated with "dirty" shadowing

        "Comet tail" reverberations
    - Kidney may be completely obscured by large amount of gas in perinephric space
    - Ring – down artifacts: air bubbles trapped in fluids
    - Top differential diagnosis include: renal calculi, nephrocalcinosis, papillary necrosis

Wan et al described two distinct types using X ray, USG, CT Abdomen,

Type I - Parenchymal destruction with streaky or mottled parenchymal gas with an absence of fluid collection. Mortality is upto sixty percent.

Type II - Renal or perirenal fluid collection with bubbly gas collection in the perinephric space or in the collecting system. Mortality is upto twenty percent.

Few images of USG KUB are shown below

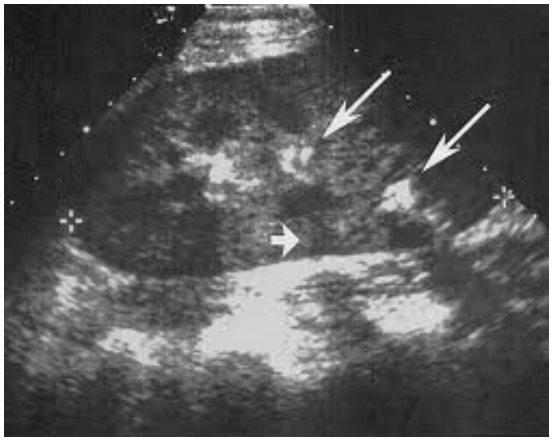


Fig - Long arrows shows multiple hyperechoic foci **S**mall arrows shows dirty acoustic shadows

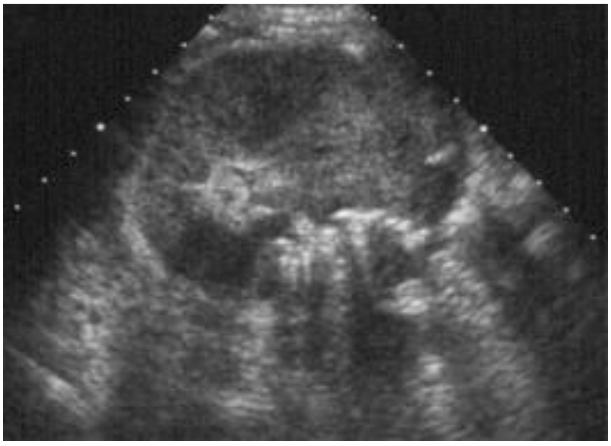


Fig – Type II disease

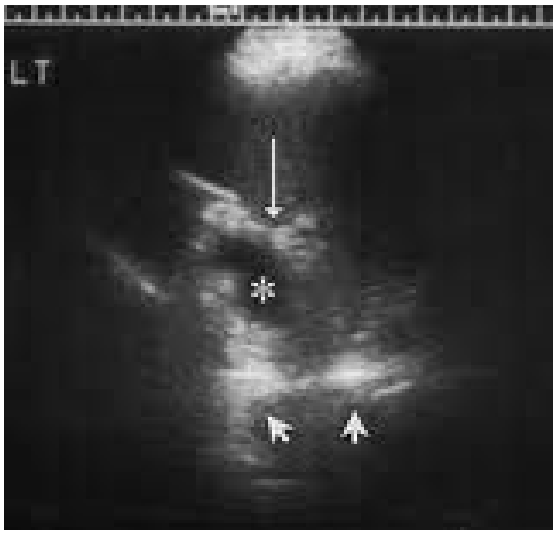


Fig - Long arrow shows high amplitude echoes represents air with fluid collection

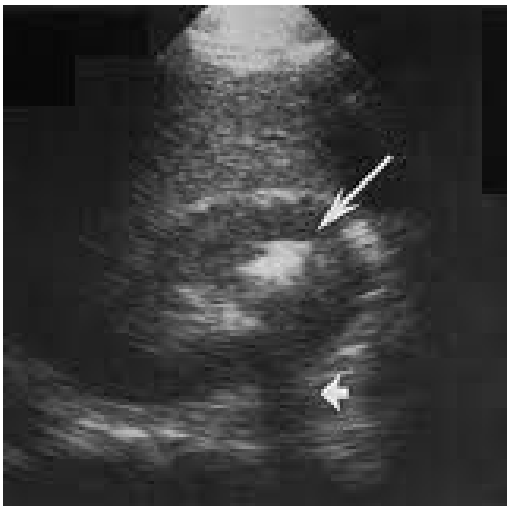


Fig – Type I disease

CT KUB

Noncontrast CT KUB is the is the most sensitive investigation for the diagnosis of this disease. It demonstrates the extent of disease, detects renal stone and points out obstruction of the urinary tract, if present.

Huang et al modified the staging proposed by Michaeli as follows

- Class I – Gas confined to the collecting system
- Class II – Gas confined to renal parenchyma
- Class III A – Perinephric extension of gas
- Class III B - Extension of gas beyond gerota fascia
- Class IV – Bilateral EPN or EPN in single functioning kidney

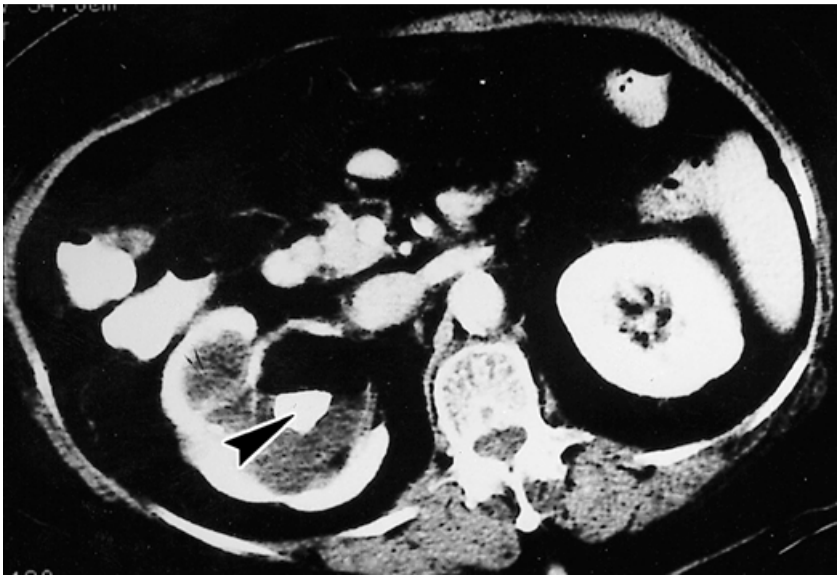


Fig – Class I disease. Gas in right renal pelvis

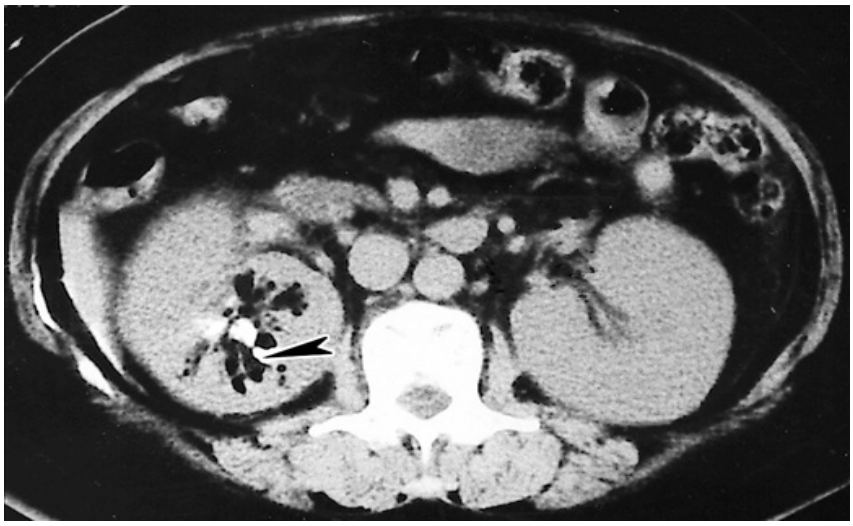


Fig – Class II disease. Gas in right renal parenchyma and right renal stone

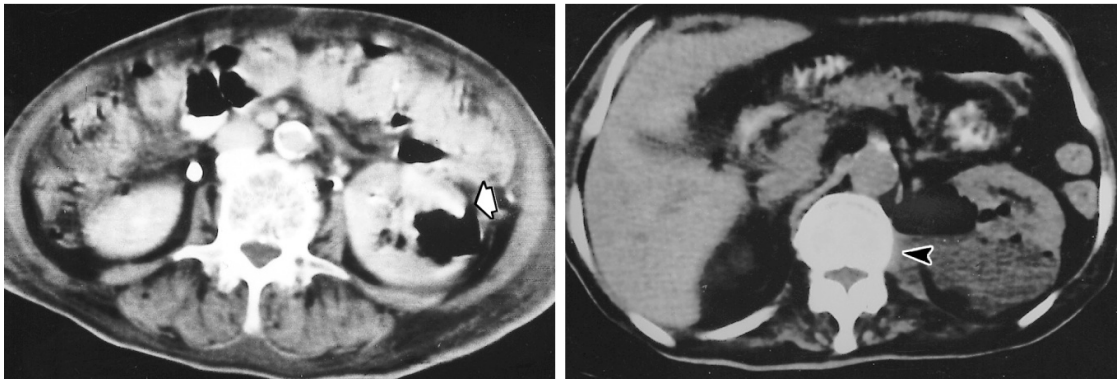


Fig – Class III disease EPN with perinephric extension and abscess formation involving left kidney

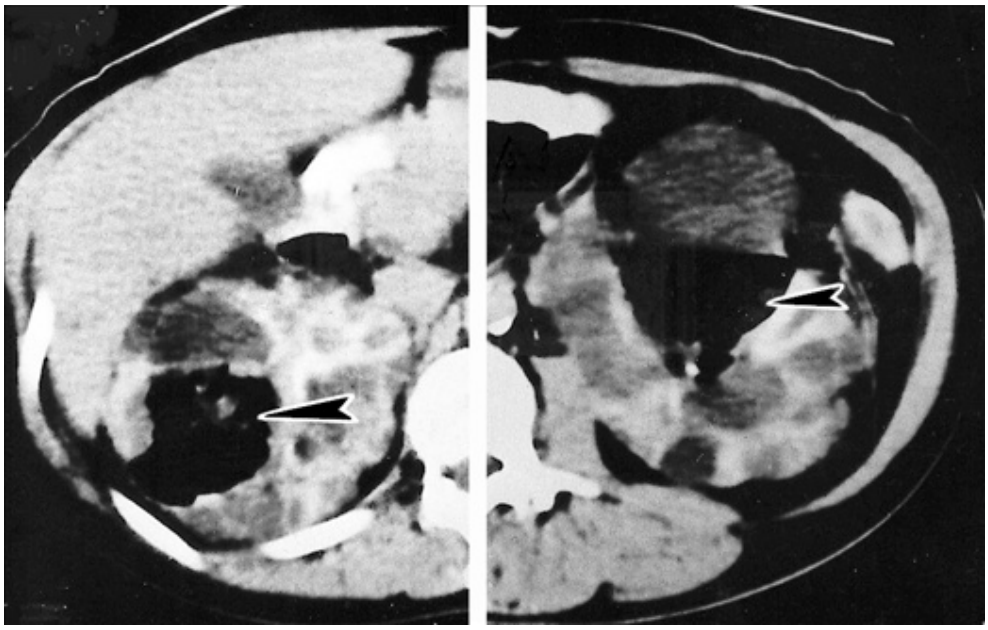


Fig – Class IV disease Bilateral EPN

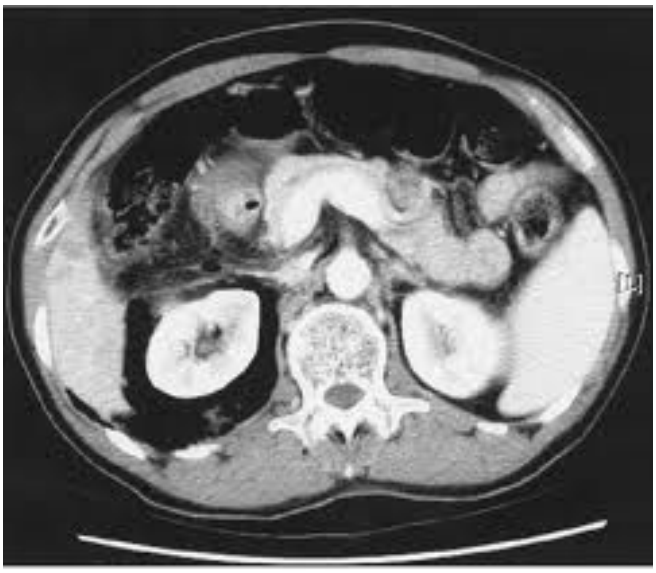


Fig – Class I disease involving right kidney

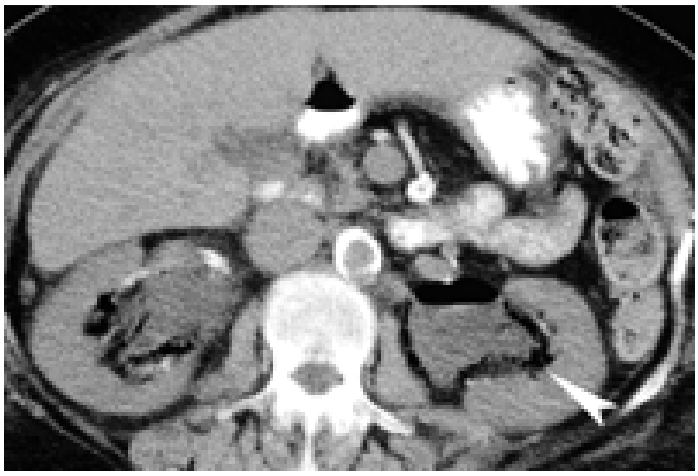


Fig – Emphysematous pyelitis with pyonephrosis and situs inversus

Thus CT KUB is not only the modality of choice for diagnosing emphysematous pyelonephritis , it is also useful in identifying renal stones and any other obstruction ,if present. It is also useful as a prognostic indicator. Class I and Class II disease carries good prognosis even if managed by medical treatment alone. Class III A and Class III B requires surgical intervention. Class IV carries worst prognosis. So CT KUB is useful in planning treatment for emphysematous pyelonephritis.

MANAGEMENT

The treatment of emphysematous pyelonephritis includes:

1. Medical treatment
2. Surgical treatment
    - Percutaneous drainage
    - Nephrectomy

Medical treatment

Patients should be stabilised before being subjected to radiological procedures. Intravenous fluids, inotropes may be needed if they present with shock and hypotension. Appropriate antibiotics should be started after taking necessary investigations . Blood, platelets may be required for treating thrombocytopenia and septicaemia. If blood sugar is elevated insulin should be started. Diabetic ketosis should be managed appropriately. Elevated renal parameters and worsening renal function may require renal replacement therapy in the form of hemodialysis. Sepsis should be aggressively treated. Most of the patients with Class I and Class II will improve with appropriate medical treatment. But Class III and Class IV patients often requried surgical intervention.

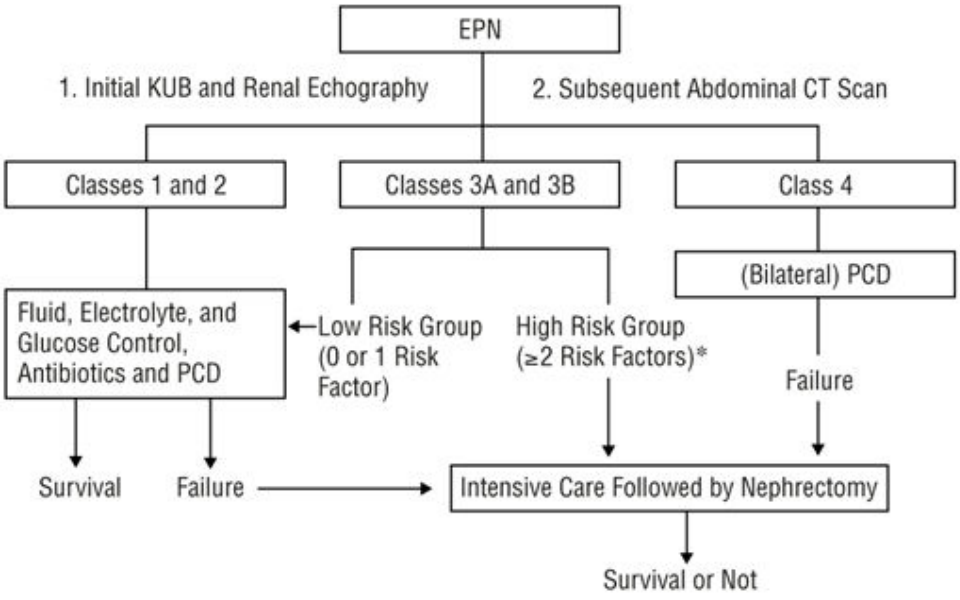
Surgical treatment

After stabilising the patient and doing CT KUB the need of surigical intervention is decided. Nowadays with the availability of potent antibiotics the role of surigcal treatment has drastically decreased. However CT guided percutaneous drainage is required in case of localised pus collection. This procedure has significantly reduced the mortality rate. Many recent studies have shown that emphysematous pyelonephritis can be sucessfully treated with antibiotics and renal replacement therapy alone. Huang et al has suggested that surgical intervention is required in late stages of emphysematous pyelonephritis. Class IV EPN is potentially fatal. But nephrectomy may be necessary in Class III and Class IV patients .

Since patients with emphysematous pyelonephritis may have associated diabetic nephropathy and chronic renal failure even after nephrectomy they may require renal replacement therapy postoperatively.

So the decision for nephrectomy should be judicially taken at the appropriatie time .

Chart - Algorithm for the management of EPN. risk factors - thrombocytopenia , shock, altered sensorium, acute renal injuriy



Prognostic factors

Poor prognostic factors are

- Age more than 60 years
- Long duration of diabetes mellitus
- Preexisting diabetic nephropathy
- Associated immuno compromised state
- Associated obstruction of urinary tract
- Previous history of instrumentation
- Poor blood sugar control
- High HbA_1C value

- ❖ Diabetic ketosis at admission
- ❖ Acute kidney injury
- ❖ Altered sensorium
- ❖ Thrombocytopenia
- ❖ Hypotension
- ❖ Comorbid illness like Coronary artery disease, hypertension
- ❖ Casuative organism – Clostridium, pseudomonas, fungal org
- ❖ Class III A and Class III B disease
- ❖ Class IV disease

Mortality with medical therapy – 60 to 70% , can be reduced to 20% with surgical drainage. Patients who presents with sepsis, shock, renal failure have higher mortality.

MATERIALS AND METHODS

1. Study design

It is a prospective and observational study.

2. Study group

All type 2 diabetic patients presenting with urinary tract infection are screened with ultrasonogram of KUB. Ultrasonogram proven emphysematous pyelonephritis are included in this study.

3. Place of the Study

Kilpauk Medical College and Hospital

4. Period of Study

January 2011 – December 2012

5. Collabarating Departments

- Nephrology department
- Diabetology department
- Radiology department
- Microbiology and biochemistry department

6. Conflict of interest

Nil

7. Inclusion and exclusion criteria

All type 2 diabetic patients (both males and females) more than eighteen years of age presenting with urinary tract infection are screened with ultrasonogram . Patients with ultrasonogram proven emphysematous pyelonephritis are included in this study. Patients who do not satisfy the above criteria are excluded from this study.

8. Consent

Informed consent is obtained from the patients before including them in the study.

9. Methodology

a. Clinical Examination

b. Investigations done

INVESTIGATIONS DONE

1. CBC with platelet count
2. Urine albumin, sugar, deposits, acetone, spot PCR
3. Urine culture and sensitivity
4. Blood culture and sensitivity
5. Blood sugar
6. Blood urea
7. Serum creatinine
8. HbA_1C
9. Antibodies for HIV1 and HIV2
10. X Ray KUB
11. USG KUB
12. CT KUB

STATISTICAL ANALYSIS

Mean values of all parameters in subgroups were calculated by independent sample-t-test. To compare the distributions of dichotomous data viz .gender, symptomatology, urine acetone, cultures for growth, need of dialysis/surgical intervention, Chi-square test was used. Association between variables was assessed by logistic regression model. Potential confounders were adjusted for.

Pearson correlations were applied to evaluate the correlation between Emphysematous pyelonephritis and age, sex, history of fever,flank pain & UTI symptoms, UT obstruction, renal stones& use of instrumentation, blood culture & mode of therapy. All statistical analyses were performed using the SPSS package .A p-value of less than 0.05 was considered to be statistically significant.

The analysis is done among various characteristics identified in a case series of emphysematous pyelonephritis.

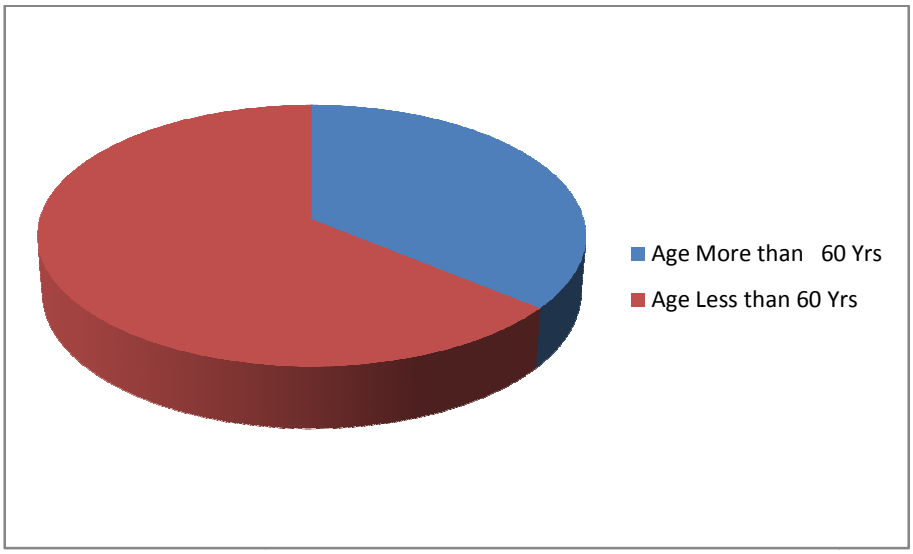
AGE DISTRIBUTION:

The age distribution of the study population is as follows:

Table 1

Age More than 60 Yrs	9
Age Less than 60 Yrs	16

Fig 1



The percentage of patients of age less than 60 years 64%

The percentage of patients of age more than 60 years 36%

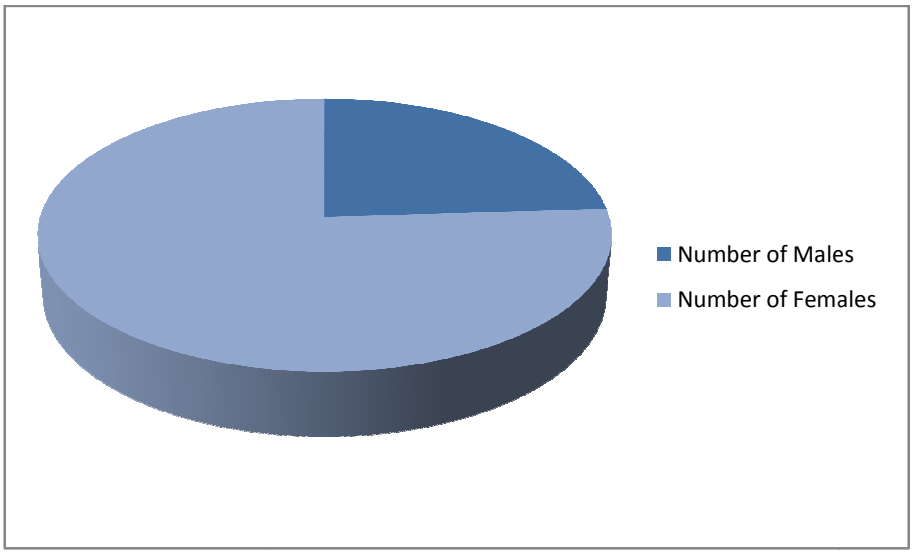
SEX DISTRIBUTION OF STUDY GROUP:

The sex distribution of the study population is as follows:

Table 2

Number of Males	6
Number of Females	19

Fig 2



The percentage of males – 24%

The percentage of females – 76%

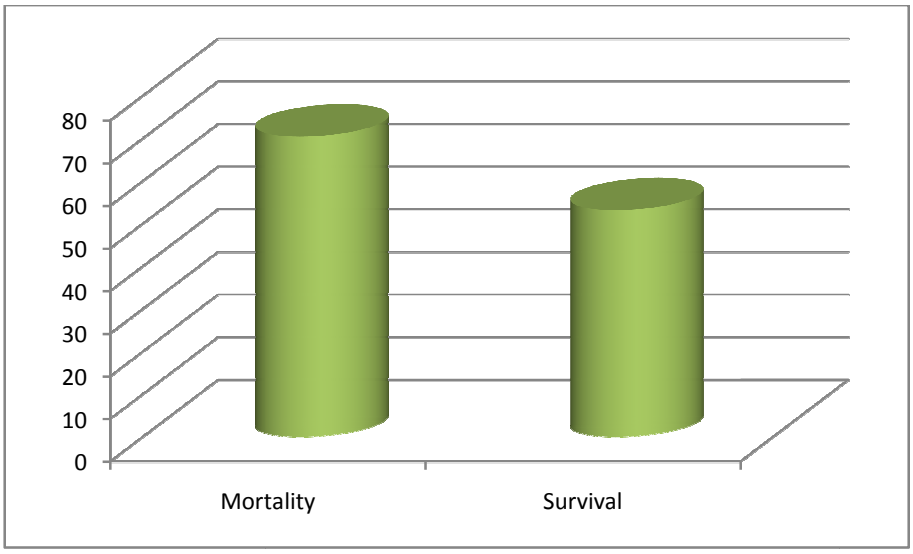
AGE

The correlation between age and mortality in the study population.

Table 3

	Out come	Mean
Age	Mortality	70.38
	Survival	53.12

Fig 3



The mean age of patients who survived is 53.12 years. The mean age of patients who died is 70.38 years . The correlation between the age of the patient and mortality outcome is **statistically significant** ($p<0.05$) i.e. higher the age of the patient, greater is the risk of mortality.

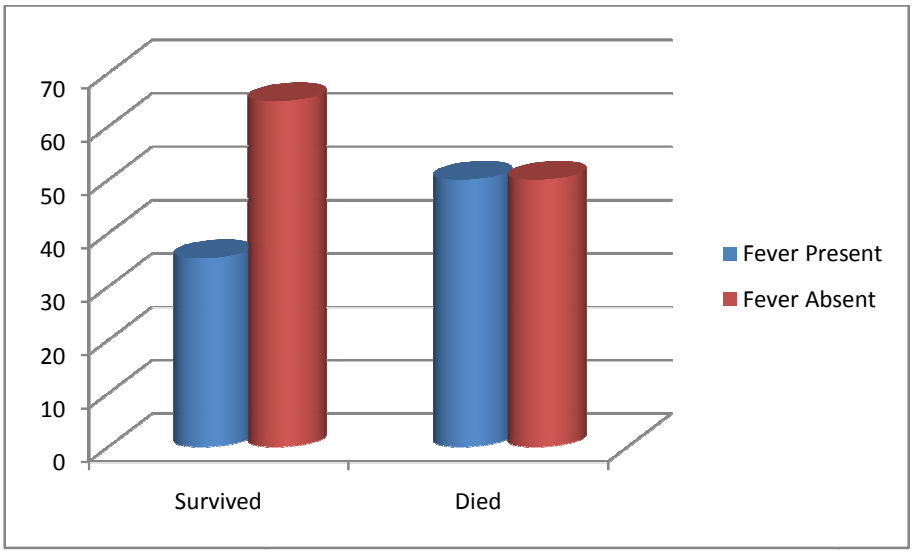
HISTORY OF FEVER

The correlation between history of fever and mortality in the study population

Table 4:

	Survival	Mortality
Fever Present	35.3	50
Fever Absent	64.7	50

Fig 4



P = 0.484

There is no statistical significance with regards to correlation between history of fever and mortality.

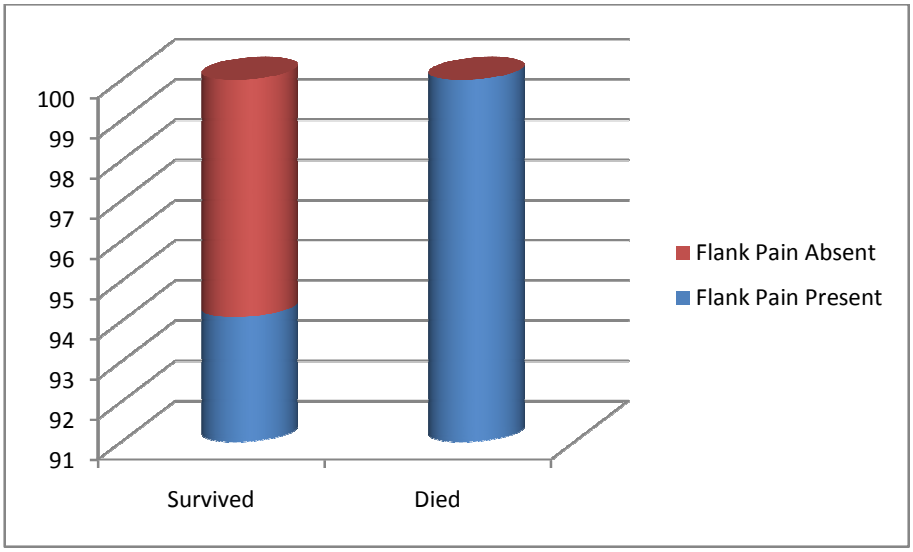
HISTORY OF FLANK PAIN

The correlation between history of flank pain and mortality in the study population

Table 5

	Survival	Mortality
Flank Pain Present	94.10	100
Flank Pain Absent	5.90	0

Fig 5



P = 0.484 Statistically not significant.

There is no statistical significance with regards to correlation between history of flank pain and mortality.

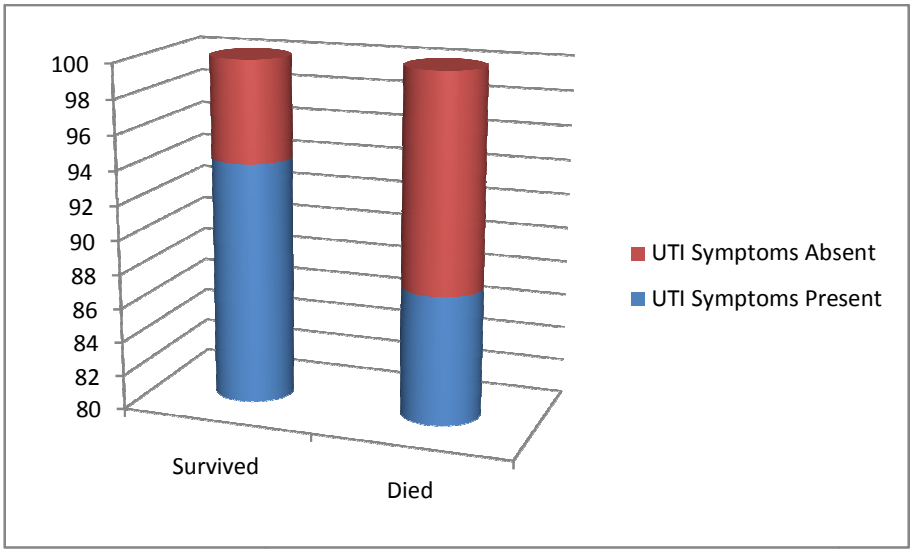
UTI SYMPTOMS

The correlation between history of UTI symptoms and mortality in the study population

Table 6

	Survival	Mortality
UTI Symptoms Present	94.1	87.5
UTI Symptoms Absent	5.9	12.5

Fig 6



P = 0.569

There is no statistical significance with regards to correlation between history of UTI symptoms and mortality

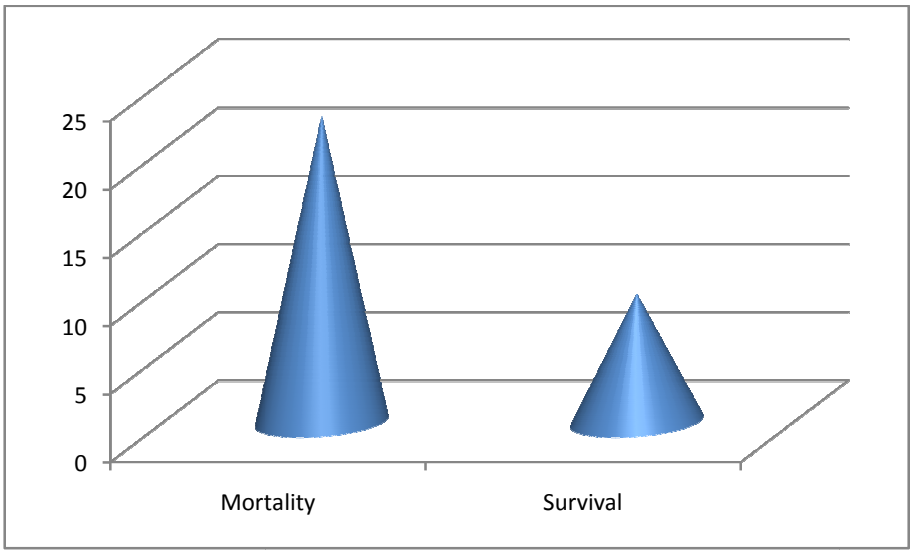
DURATION OF DM

The correlation between duration of diabetes mellitus and mortality

Table 7

	Out come	Mean
Duration of DM	Mortality	22.38
	Survival	9.35

Fig 7



There exist a **statistically significant** P = 0.000(< .05) correlation between duration of diabetes mellitus and mortality. The mean duration of DM in years in patients who survived is 9.35. The mean duration of DM in years in patients who died is 23.38.

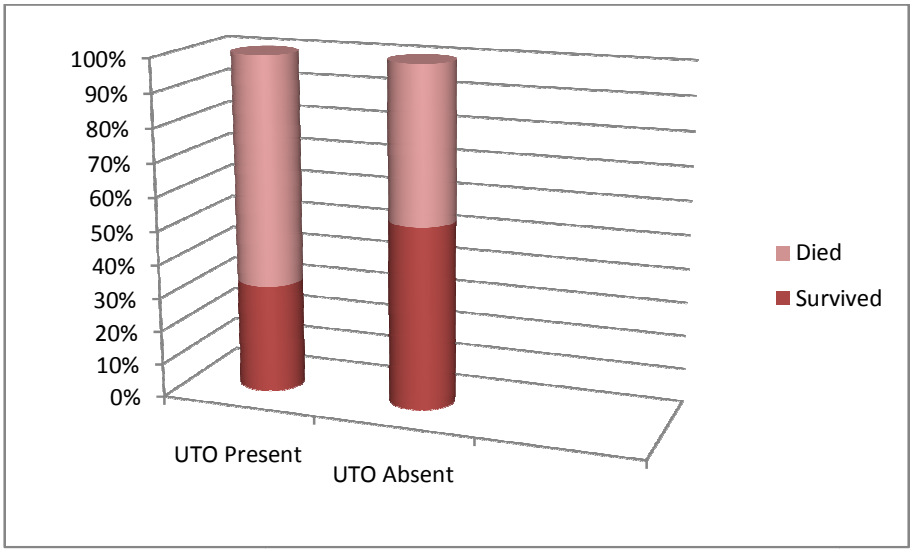
HISTORY OF URINARY TRACT OBSTRUCTION

The correlation between history of urinary tract obstruction and mortality in the study population.

Table 8

	Survival	Mortality
UTO Present	11.8	25
UTO Absent	88.2	75

Fig 8



P = 0.400 The correlation between history of urinary tract obstruction and mortality in the study population is not statistically significant.

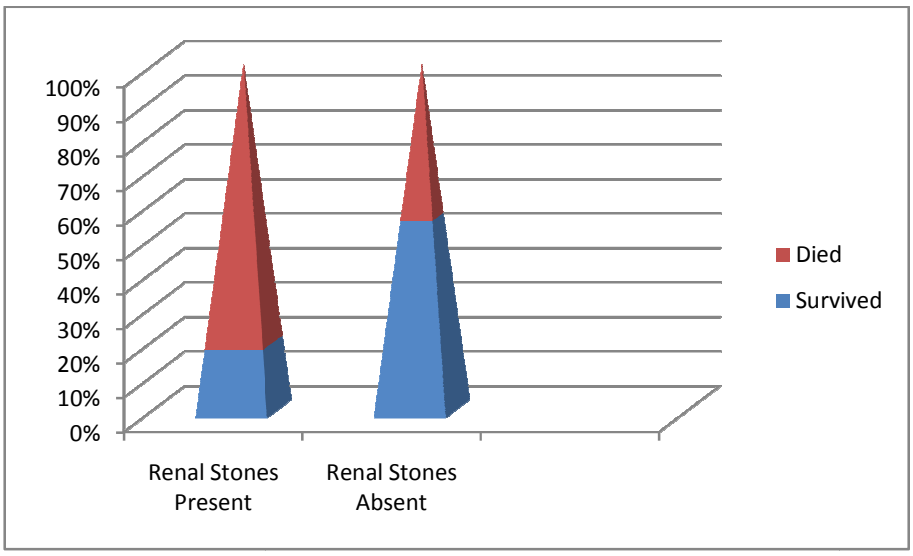
HISTORY OF RENAL STONES

The correlation between renal stones and mortality in the study population

Table 9

	Survival	Mortality
Renal Stones Present	5.9	25
Renal Stones Absent	94.1	75

Fig 9



P = 0.170 Statistically not significant.

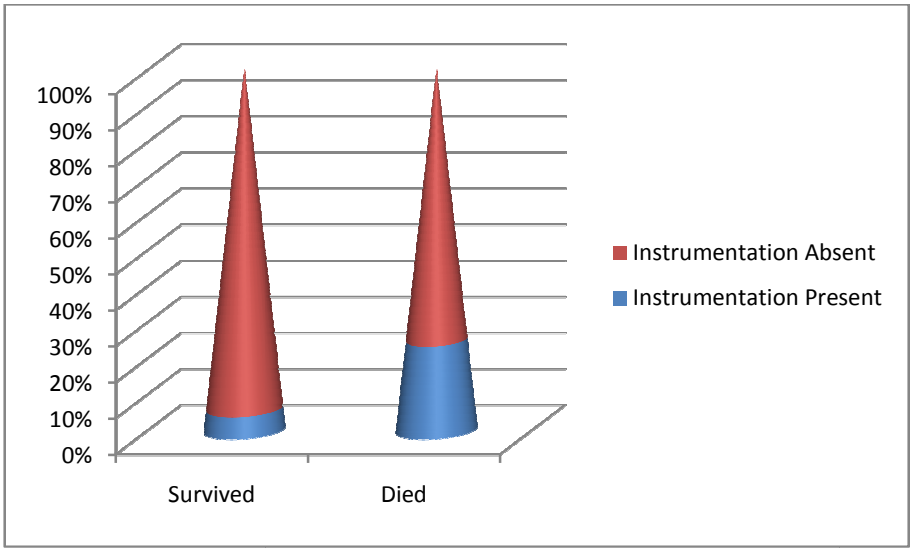
HISTORY OF INSTRUMENTATION

The correlation between history of instrumentation and mortality in the study population.

Table 10

	Survival	Mortality
Instrumentation Present	5.9	25
Instrumentation Absent	94.1	75

Fig 10



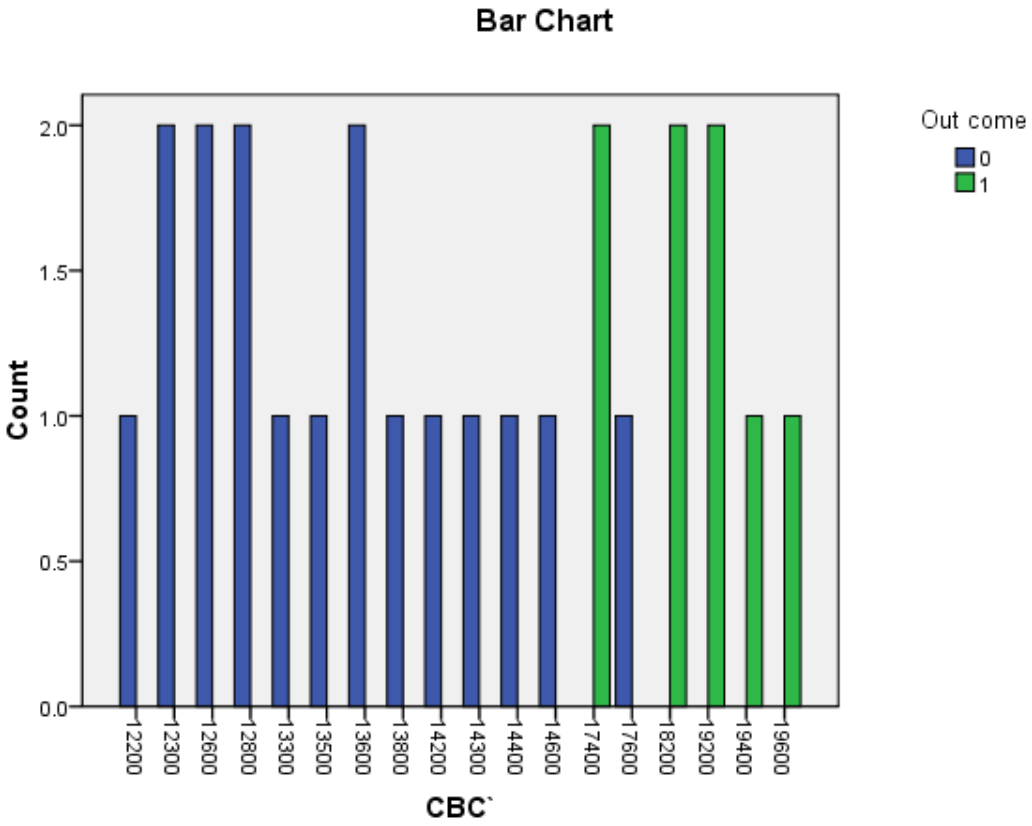
P = 0.170 The correlation between history of instrumentation and mortality in the study population is not statistically significant.

COMPLETE BLOOD COUNT

The correlation between complete blood count and mortality in the study population

Table 11

	Out come	Mean
CBC	Mortality	18575.00
	Survival	13558.82



P = 0.000 (< 0.05) **Statistically Significant**

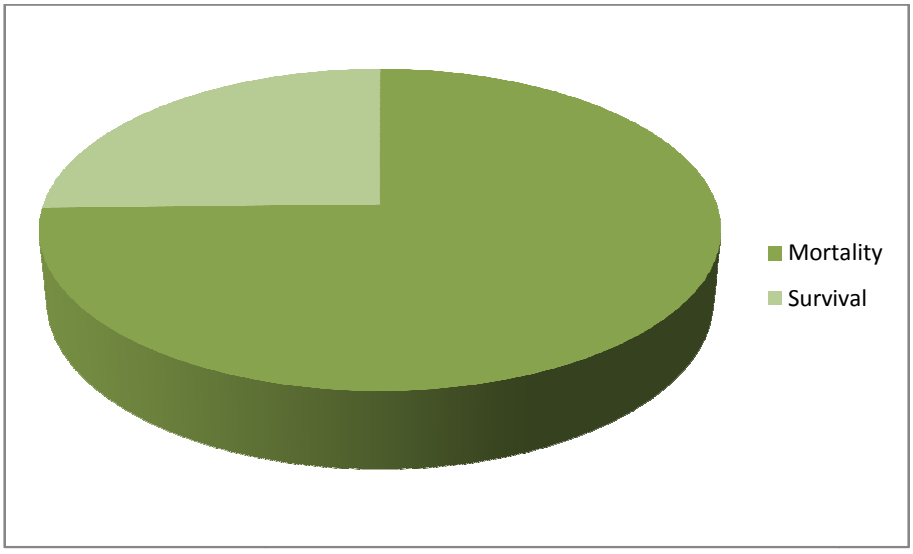
PLATELET COUNT

The correlation between platelet count and mortality in the study population

Table 12

	Out come	Mean
Platelet count	Mortality	54000.00
	Survival	18304.81

Fig 12



There exist a **statistically significant** P = 0.000 (< 0.05) correlation between platelet count and mortality. The mean platelet count in patients who survived is 18304.81. The mean platelet count in years in patients who died is 54000.

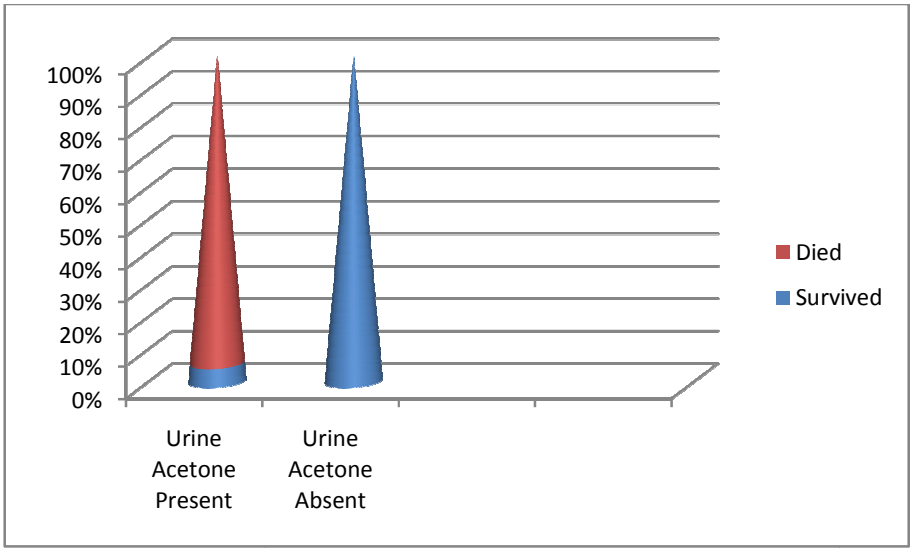
URINE ACETONE

The correlation between urine acetone and mortality in the study population

Table 13

	Survival	Mortality
Urine Acetone Present	5.9	100
Urine Acetone Absent	94.1	0

Fig 13



There exist a **statistically significant** P = 0.000 (< 0.05) correlation between urine acetone and mortality**.**

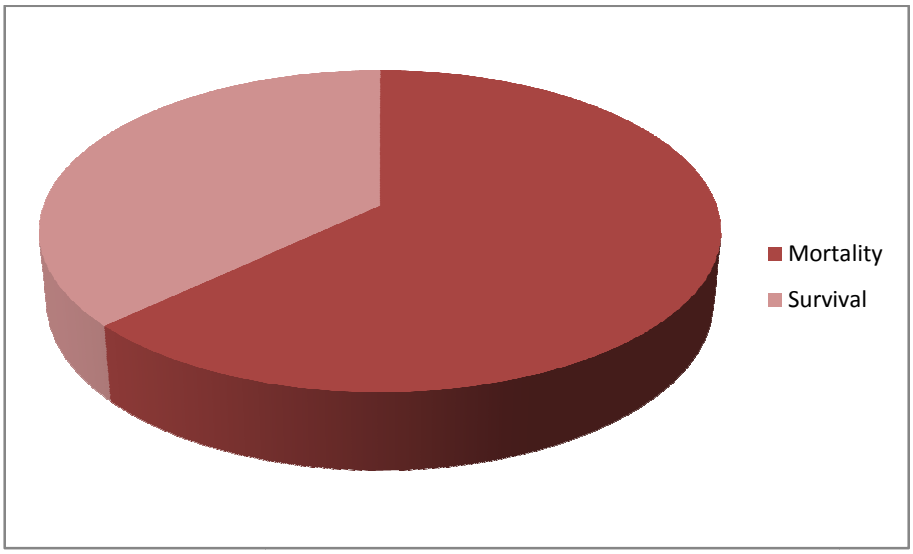
URINE SPOT PCR

The correlation between urine spot PCR and mortality in the study population

Table 14

	Out come	Mean
Urine Spot PCR	Mortality	4.52
	Survival	2.65

Fig 14



There exist a **statistically significant** P = 0.000 (< 0.05) correlation between urine acetone and mortality**.** The mean Urine spot PCR value in patients who survived is 2.65. The mean Urine spot PCR value in patients who died is 4.52.

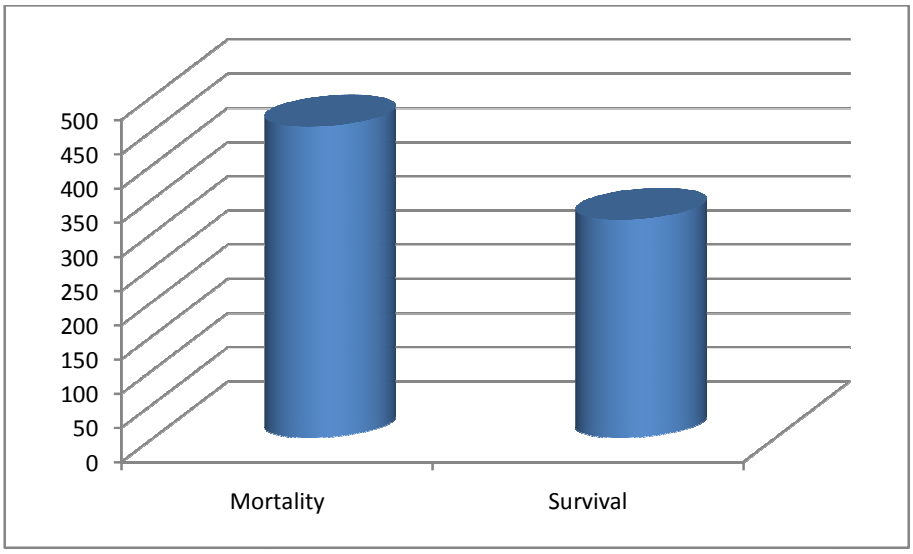
BLOOD SUGAR

The correlation between blood sugar and mortality in the study population

Table 15

	Out come	Mean
Blood Sugar	Mortality	454.25
	Survival	318.24

Fig 15



There exists a s**tatistically significant** P = 0.000 (< 0.05) correlation between blood sugar values and mortality**.** The mean blood sugar in patients who survived is 318.29.The mean blood sugar in patients who died is 454.25.

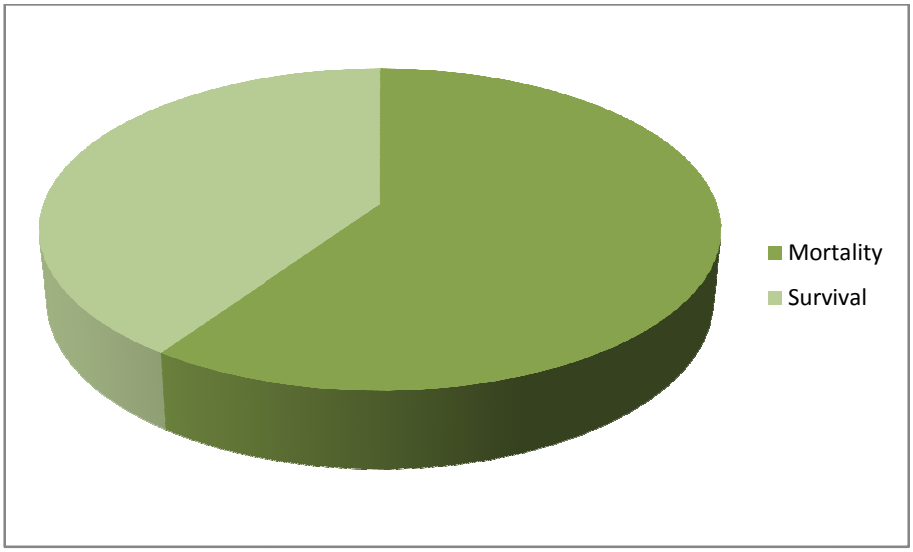
SERUM CREATININE

The correlation between serum creatinine and mortality in the study population

Table 16

	Outcome	Mean
Se Cr	Mortality	6.99
	Survival	4.74

Fig 16



P = 0.386 The correlation between serum creatinine and mortality is not statistically significant. The mean serum creatinine in patients who survived is 4.74.The mean serum creatinine in patients who died is 6.99.

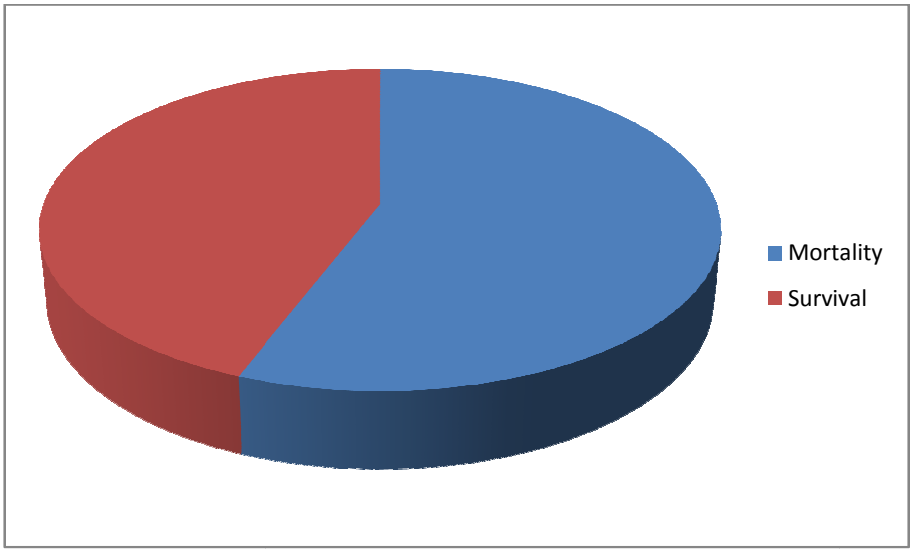
HbA$_1$C

The correlation between HbA_1C and mortality in the study population

Table 17

	Out come	Mean
HbA1C	Mortality	9.84
	Survival	7.78

Fig 17



The correlation between HbA_1C and mortality is s**tatistically significant** P = 0.000 (< 0.05) . The mean HbA_1C in patients who survived is 7.78. The mean HbA_1C in patients who died is 9.84.

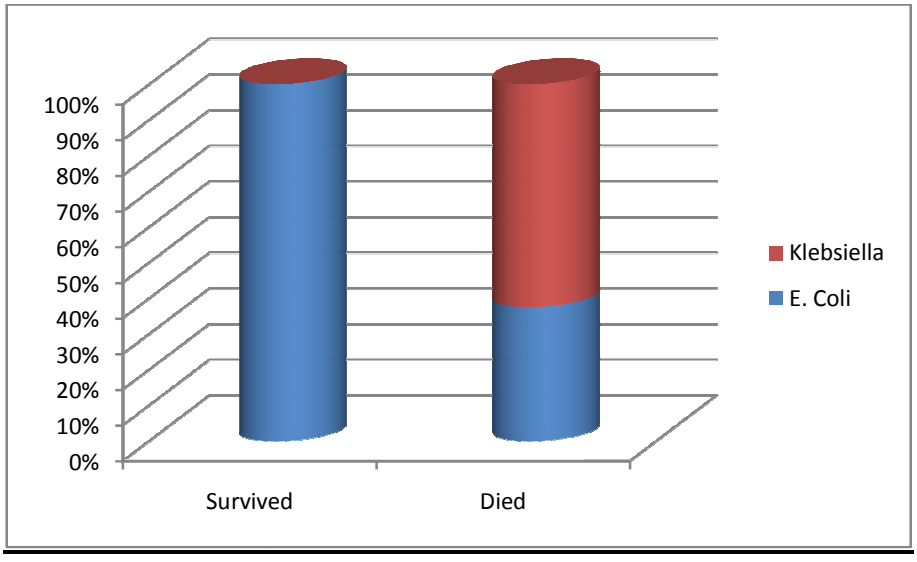
URINE CULTURE AND SENSITIVITY

The correlation between urine culture and sensitivity and mortality in the study population

Table 18

	Survived	Died
E. Coli	100	37.5
Klebsiella	0	62.5

Fig 18



The correlation between urine culture and sensitivity and mortality is **statistically significant** P = 0.000 (< 0.05)

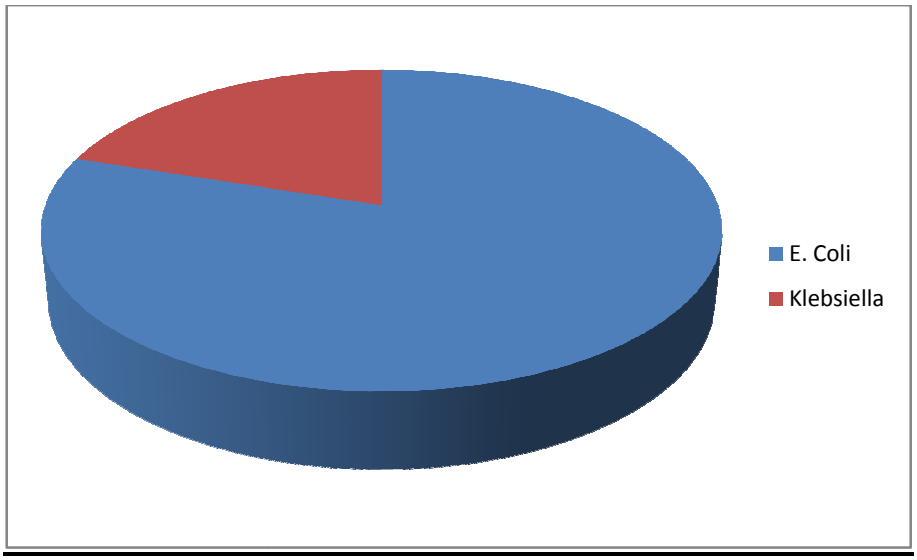
URINE CULTURE AND SENSITIVITY

The organisms grown in urine culture and sensitivity in our study population.

Table 19

E. Coli	20
Klebsiella	5

Fig 19



In 80% of cases E.Coli is grown .

In 20% of cases Klebsiella is grown.

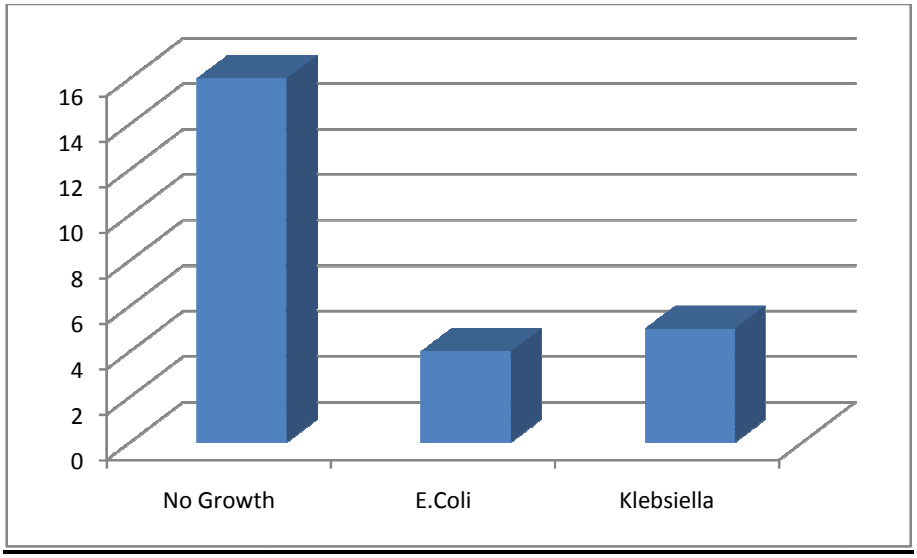
BLOOD CULTURE AND SENSITIVITY

The organism grown in blood culture and sensitivity in our study population.

Table 20

No Growth	16
E.Coli	4
Klebsiella	5

Fig 20



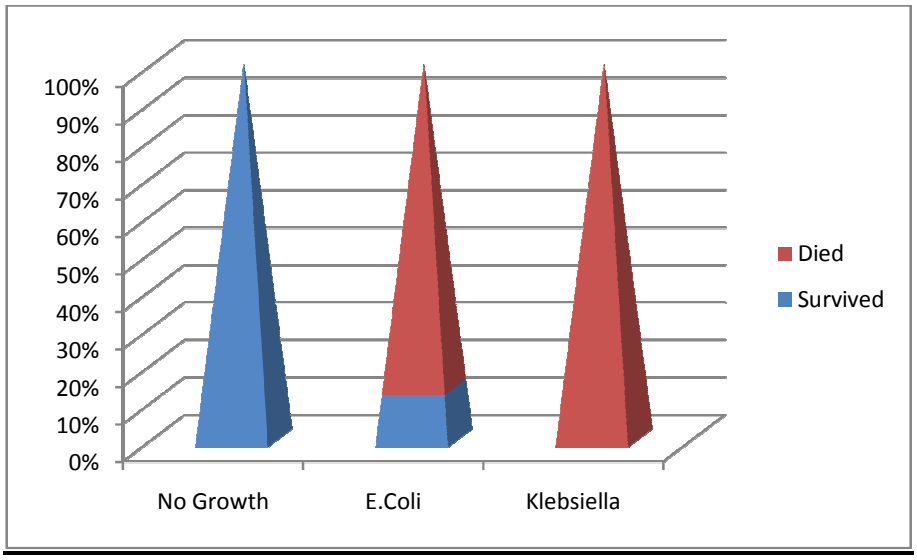
BLOOD CULTURE AND SENSITIVITY

The correlation between blood culture and sensitivity and mortality in the study population

Table 21

	Survival	Mortality
No Growth	94.1	0
E.Coli	5.9	37.5
Klebsiella	0	62.5

Fig 21



The correlation between blood culture and sensitivity and mortality is **statistically significant** P = 0.000 (<0.05)

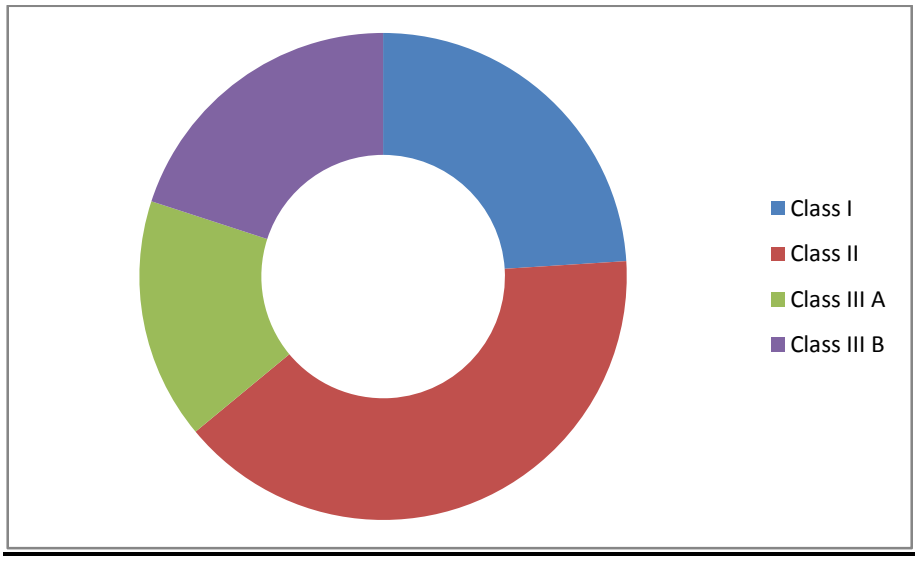
GRADING BY CT KUB

The distribution of patients in the study population according to grading by CT KUB

Table 22

Class I	6
Class II	10
Class III A	4
Class III B	5

Fig 22



The percentage of patients with Class I - 24% The percentage of patients with Class II – 40% The percentage of patients with Class III A – 16% The percentage of patients with Class III B – 20%

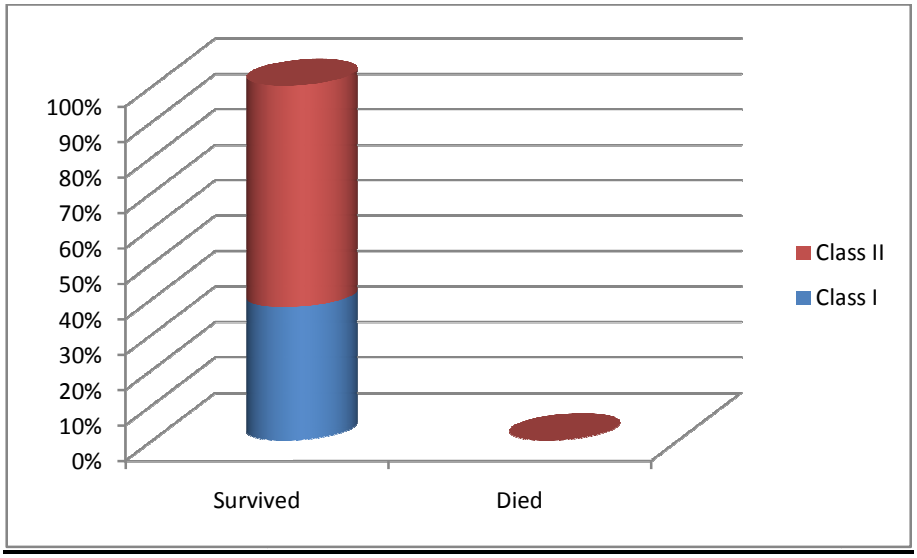
GRADING BY CT KUB

The correlation between grading by CT KUB and mortality in the study population

Table 23

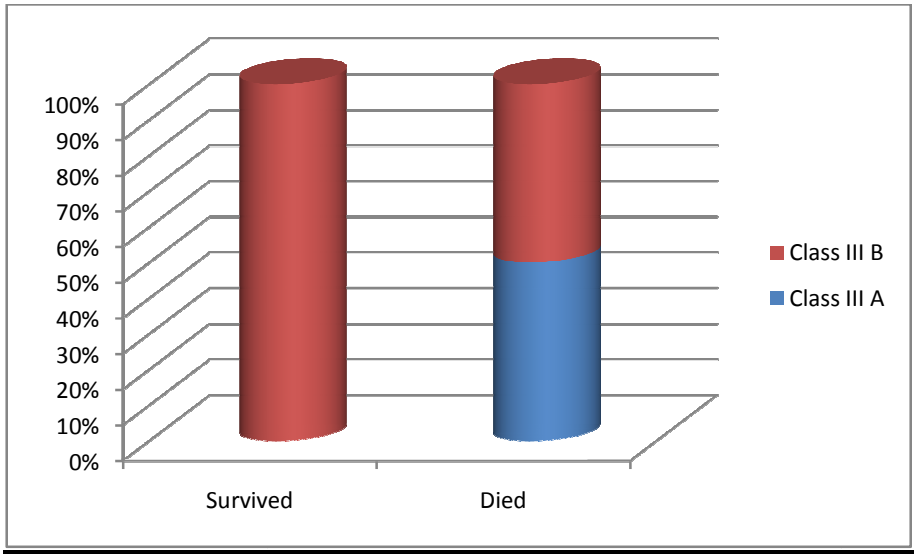
	Survival	Mortality
Class I	35.3	0
Class II	58.8	0
Class III A	0	50
Class III B	5.9	50

Fig 23



GRADING BY CT KUB

Fig 24



There exists a **statistically significant** correlation between grading by CT KUB and mortality P = 0.000 (<0.05).

MEDICAL TREATMENT

The distribution of patients who received medical treatment

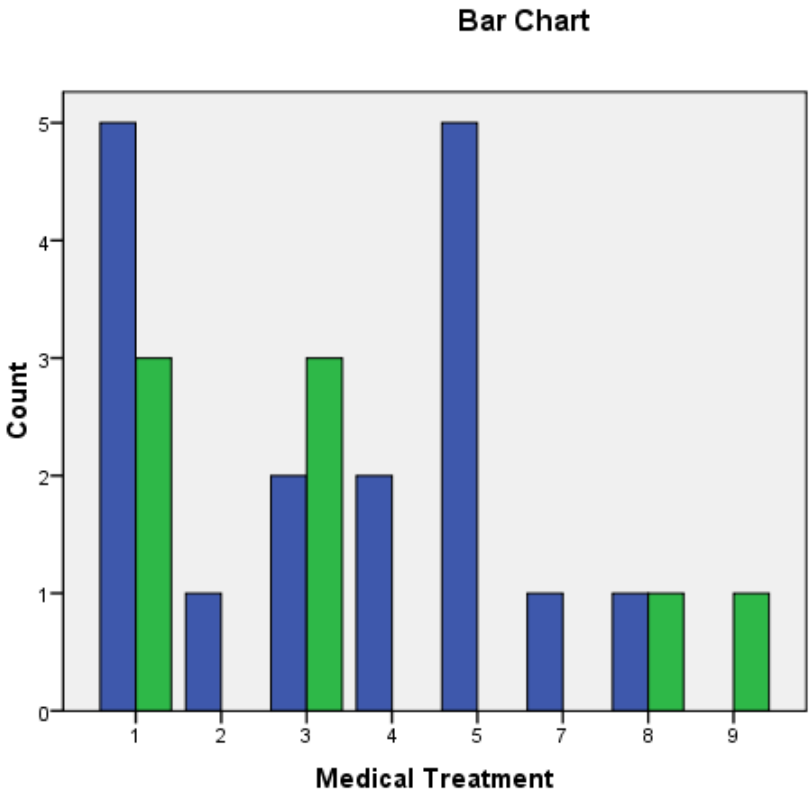


Fig 25

1. Meropenem
2. Ceftriaxone
3. Ceftriaxone and Piperacillin and tazobactam
4. Imepenem
5. Piperacillin and tazobactam
6. Vancomycin
7. Amikacin
8. Meropenem and Ceftriaxone
9. Meropenem and Vancomycin

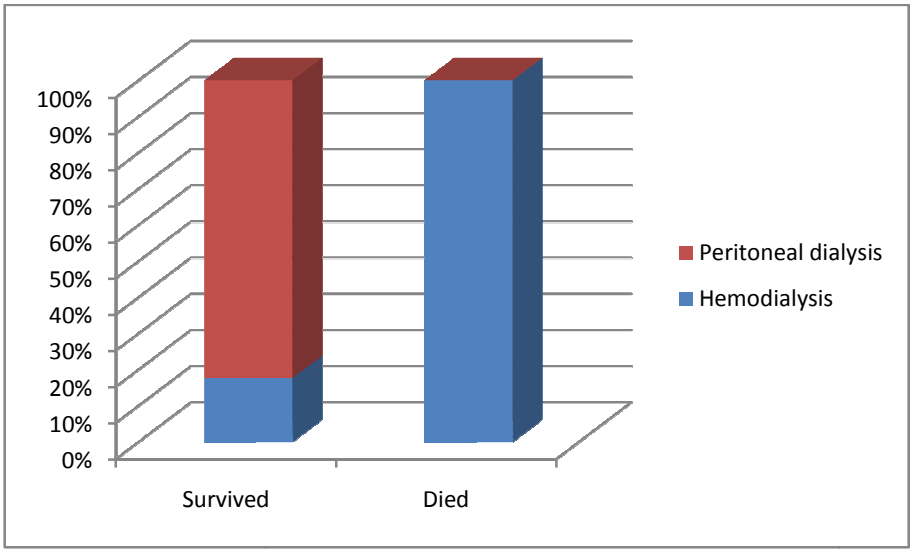
DIALYSIS

The correlation between dialysis and mortality in the study population

Table 24

	Survival	Mortality
Hemodialysis	17.6	100
Peritoneal dialysis	82.4	0

Fig 26



There exists a **statistically significant** correlation between dialysis and mortality P = 0.000 (<0.05) .

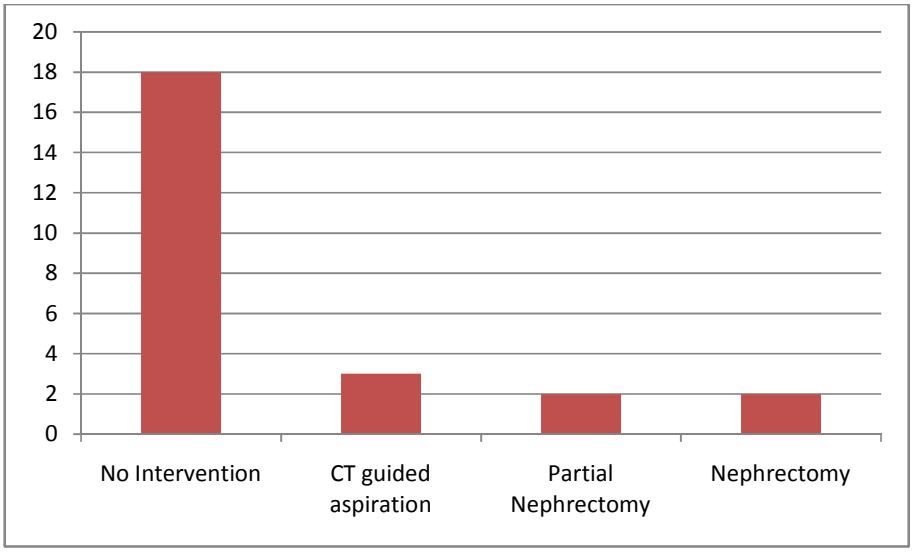
SURGICAL TREATMENT

The distribution of patients who received surgical treatment

Table 25

No Intervention	18
CT guided aspiration	3
Partial Nephrectomy	2
Nephrectomy	2

Fig 27



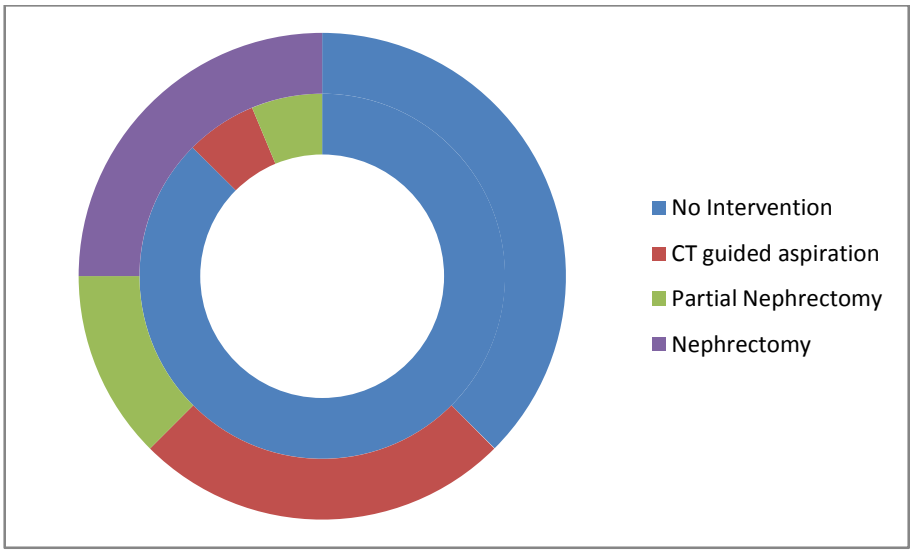
SURGICAL TREATMENT

The correlation between surgical treatment and mortality

Table 26

	Survival	Mortality
No Intervention	82.2	37.5
CT guided aspiration	5.9	25
Partial Nephrectomy	5.9	12.5
Nephrectomy	0	25

Fig 28



There exists a statistically significant correlation between surgical treatment and morality P = 0.043 (<0.05)

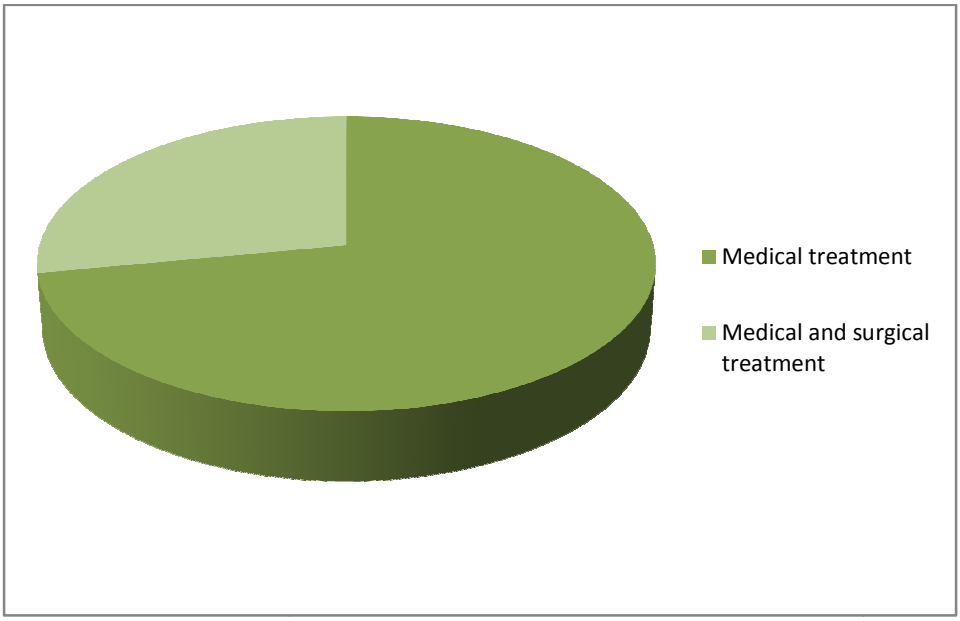
TREATMENT SUMMARY

The distribution of patients who received medical treatment alone and combined surgical treatment with mortality

Table 27

Medical treatment	18
Medical and surgical treatment	7

Fig 29



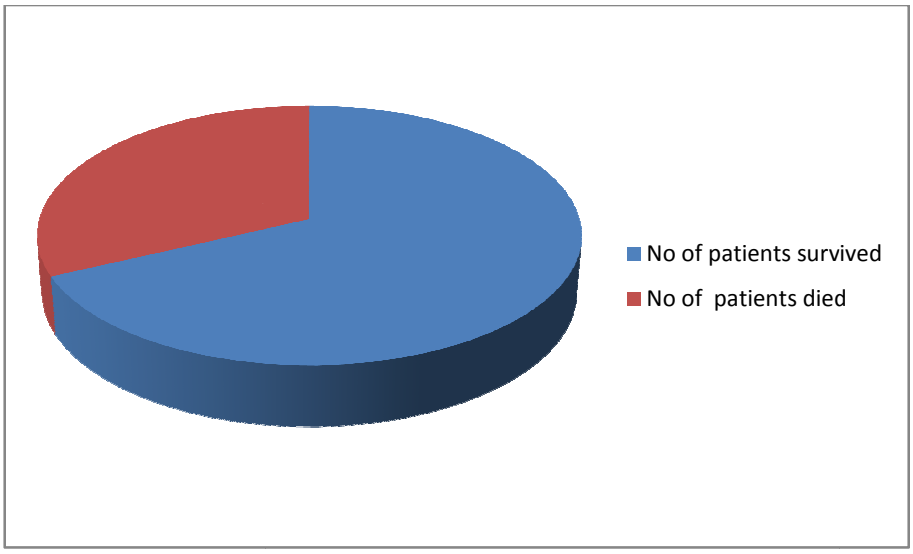
SURVIVAL AND MORTALITY

The correlation between surgical treatment and mortality

Table 28

No of patients survived	17
No of patients died	8

Fig 30



DISCUSSION

In our study it is found that, 24% of the patients are above sixty years of age, 76% of patients are below sixty years of age. All the above sixty years of age has severe disease and did not survive. The mean age of patients who survived is 53.12 years . The mean age of the patients who died is 70.38 years. In a previous study the mean age of the patients was shown to be 58 years. In that study, the mean age of patients who survived was 59 years . The mean age of the patients who died was 67 years.

In our study the ratio between affected female and male is 3.16:1. So there is female preponderance in our study. In a previous study the female: male ratio was 5.6:1.

Although the classical symptoms of this disease are fever and flank pain, urinary tract symptoms, in our study fever is not present in 32% of our patients. UTI symptoms are not present 4%. In a previous study fever is not present in 21% of patients.

In our study Urinary tract obstruction is present in 16% of the patients. 8% of them have urinary stricture and 8% have benign prostatic

hypertrophy. 8% of the patients have undergone transurethral resection of prostate. In a previous study, it was shown that 24% of the patients had urinary tract obstruction.

In our study, history of instrumentation is present in 12% of patients. 8% of the patients have renal stones. In a previous study, history of instrumentation is noted in 20% of the patients.

In our study, the mean duration of diabetes mellitus is 16 years. The mean duration of diabetes mellitus in patients who survived is 9.3 years. The mean duration of diabetes who died is 22.3 years . The mean duration of diabetes mellitus in a previous study was about 20 years.

In our study, leukocytosis is present in 95% of the patients. The mean total count in patients who survived is 13558. The mean total count in patients who died is 18575. In a previous study 69% of the patients had leukocytosis.

In our study, thrombocytopenia is present in 70% of the patients. The mean platelet count in patients who survived is 18304. The mean platelet count in patients who died is 54000. In a previous study 48% of

the patient had thrombocytopenia . In that study, 36% of the patients with thrombocytopenia had survived and 64% of the patients died.

In our study, 32% of the patients have positive urine acetone during admission. All the patients with diabetic ketosis died.

In our study, proteinuria is present in 85% of the patients. In a previous study, proteniuria was present in 20% of the patients.

In our study, the mean blood sugar in patients who survived is 318.24. The mean blood sugar in patients who died is 454.25. 87% of the patients who had diabetic ketosis at admission required surgical intervention. The mortality in patients with diabetic ketosis is 100%.

In our study, the mean serum creatinine in patients who survived is 4.74% and died is 6.99% . In a previous study 28% of the patients with elevated creatinine had died and 67% of the patients had survived.

In our study the mean HbA_1C in patients who survived is 7.78. The mean HbA_1C who died is 9.84 . 85% of the patients had higher HbA_1C. In a previous study, 70% of patients had higher HbA_1C.

In our study E.coli is grown in urine culture among 80% of the patients. Klebsiella is grown in 20% . The mortality is 100% in patients who had growth of Klebsiella and 15% in E.coli. All the patients who had Klebsiella grown also had blood culture positive for Klebsiella.

All the patients with growth of Klebsiella required surgical intervention. There is no mixed culture positivity in our study. In a previous study it is shown that E.coli is grown in 70% of the patients and Klebsiella in 28% of the patients.

In our study, in blood culture 20% of the patients had Klebsiella grown. E.coli is grown in 16% of the patients. No organism is grown in 64% of the patients. The mortality rates in patients with Klebsiella is 100% and E.coli is 16%. There is no mixture culture positivity.

In our study, as we grade the disease by CT KUB, Class I disease is present in 24% of the patients and Class II in 40% of the patients. All the patients who had Class I and Class II disease survived. Class III A and Class III B is present in 16% and 20% respectively. The mortality rate in Class III A and B is 75% and 100% respectively. All the patients in Class III A and B requires surgical intervention. In a previous study, the

mortality rate in patients with Class I and Class II is 0 and 10% respectively. The mortality rate in patients with Class III A and B is 29% and 9% respectively. The mortality rate in Class IV disease is 50%.

In our study, all the patients require renal replacement therapy. 56% of the patients required peritoneal dialysis. All those patients who required peritoneal dialysis survived. 44% of the patients required hemodialysis. The mortality rate in patients treated with hemodialysis is 72%.

In our study, all the patients are treated with appropriate antibiotic according to sensitivity pattern. 72% of the patients does not require any surgical intervention. The mortality rate of patients who are treated medical therapy alone is 11%. But all of them belong to Class I and Class II group.

In our study, 28% of the patients required surgical therapy. 72% required medical treatment alone. CT guided aspiration is done in 12% of the patients. 16% of the patients required nephrectomy. The mortality rate of the patients who underwent nephrectomy is 75%. But all of them

belong to Class III A and B groups. All the patients in Class III A and B requires surgical intervention

In a previous study, the over all mortality rate is 32%. In the patients who died surgical intervention is done in 75% of patients. 25% of the patients were given only medical treatment(antibiotics). In a previous study, the mortality rate in patients who are treated with antibiotics alone is 38%. The mortality rate in patients with surgical intervention is 60%

CONCLUSION

To summarise the results of our study

- Female patients are more commonly affected than males
- Mortality due to the disease is greater in patients above sixty years of age.
- Urinary tract obstruction is a separate risk factor for mortality in addition to Diabetes mellitus.
- Mortality rate is more if the duration of diabetes is more than sixteen years.
- Thrombocytopenia at presentation increases mortality rate.
- All the patients who are dmitted with diabetic ketosis succumb to the illness.
- Patients with poor glycemic control reflected by high HbA_1C carried poor prognosis.
- The commonest organism grown in urine culture is E.coli. Klebsiella is grown in 20% of the patient.
- All patients with klebsiella grown in urine culture also have blood culture positive for klebsiella.
- The mortality rate is 100% in klebsiella grown patients, when compared to E.coli (15%)

- 36% of the patients have sterile blood culture.
- The mortality rate is high in patients who had blood culture positivity. (Klebsiella - 100%, E.coli – 75%)
- 64% of patients had Class I, Class II disease (as per CT KUB), all of them survived with medical treatment alone.
- The mortality rate is 75% in Class III A and 100% in Class IIIB
- All the patients required renal replacement therapy.
- 72% of patients are treated by medical therapy alone, in which the mortality rate is 11% .
- 28% of patients required surgical intervention , 12% were treated with CT guided aspiration and 16% with nephrectomy. The mortality rate in patients with nephrectomy 75%

As per the inference from our study

E.coli or Klebsiella infection in patients with diabetes mellitus is the corner stone for development of EPN. Emphysematous pyelonephritis can be successfully as follows.

- Although surgery was performed routinely, consensus now favors conservative therapy including placement of catheter drainage nephrostomy for drainage.
- All patients are treated with antibiotics and obstruction, if present should be relieved.
- Class I disease can be treated with antibiotics alone.
- Class II disease can be treated with antibiotics and percutaneous catheter drainage, if necessary.
- Class III A and III B disease at low risk can be initially treated with antibiotics and percutaneous catheter drainage. Sometimes nephrectomy may be necessary.
- Class III A and III B disease at high risk require antibiotics plus nephrectomy.
- Class IV disease should initially be treated with antibiotics and percutaneous catheter drainage. Nephrectomy is a last option.
- Nephrectomy is indicated in all patients in whom percutaneous catheter drainage is unsuccessful.

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PROFORMA

Personal data

Name: Age: Sex:

Address: Occupation: I.P No:

Date of Admission: Date of Discharge:

History

1. Fever
2. Abdominal Pain, Flank Pain
3. Vomiting
4. Urinary Symptoms
5. Other Symptoms

Past History

History of Diabetes Mellitus

- Duration
- Mode of Therapy
- Control Status

History of Comorbid Illness

- Hypertension
- Coronary Artery Disease

Previous Hospitalisation

History of Urinary tract obstruction

History of Renal Stones

History of Instrumentation, trauma

History of Immunosuppressive illness, drugs

Family History

1. Diabetes Mellitus

2. Other Illness

Personal History

Whether Smoker, Alcoholic

Clinical Examination

Height Weight

Vitals

Pulse Rate Respiratory Rate

Blood Pressure Temperature

Systemic examination

Abdomen

1. Inspection
2. Palpation
3. Percussion
4. Auscultation

CVS

RS

CNS

Investigation

1. CBC

2. Urine Albumin Sugar Spot PCR
   Deposit Acetone

3. Urine Culture and sensitivity
   1 2 3

4. Blood Culture and Sensitivity

5. Blood urea and Serum Creatinine

6. Hb1AC

7. Antibodies for HIVI and HIV2

8. X-RAY KUB

9. USG KUB

10. CT KUB – grading

Treatment

1. Fluids

2. Antibiotics

3. Dialysis – Peritoneal Dialysis/ Hemodialysis

4. Percutaneous drainage/ CT guided drainage/ Stenting

5. Nephrectomy

MASTER CHART

SL NO	Patient's Name	Age	Sex	H/o Fever	Flank Pain	Vomiting	UTI Symptoms	H/O DM	Duration of DM	H/o Urinary tract obstruction	H/o of Renal Stones	H/o Instrumentation	CBC`	Platelet count	Urine Deposits Pus cells	Urine Acetone	Urine Spot PCR	Urine C/S	Blood C/S	Blood Sugar	Se Cr	HbA1C	HIV	Xray KUB	Evidence of EPN in USG Abd	Grading of EPN by CTKUB	Medical Treatment	Dialysis	Surgical Treatment	Outcome
1	Nagammal	50	F	Y	Y	Y	Y	Y	9Yrs	N	N	N	13600	178000	5	Nil	1.2	E.Coli Grown	No Growth	250	1.8	7.4	Neg	NAD	Y	Class II	Piperacillin plus tasobactam and Insulin	Peritoneal Dialysis	Nil	Survived
2	Chandra	66	F	N	Y	Y	Y	Y	22Yrs	Y	Y	N	17400	69000	15	Pos	4.5	E.Coli Grown	E.Coli Grown	440	5.8	9.2	Neg	NAD	Y	Class IIIA	Meropenem and Insulin	Hemo Dialysis	Partial Nephrectomy of right kidney	Died
3	Radhammal	46	F	Y	Y	Y	Y	Y	7Yrs	N	N	N	14300	189000	5	Nil	2.7	E.Coli Grown	No Growth	280	2.7	8.6	Neg	NAD	Y	Class II	Impeinen and Insulin	Peritoneal Dialysis	Nil	Survived
4	Swamyk annu	65	M	Y	Y	Y	Y	Y	30Yrs	N	N	N	17600	42000	10	Pos	4.7	E.Coli Grown	E.Coli Grown	456	4.7	9.9	Neg	NAD	Y	Class IIIB	Meropenem, Ceftriaxone and Insulin	Hemo Dialysis	CT guided drainage	Died
5	Ponnammal	48	F	Y	Y	Y	Y	Y	7Yrs	N	N	N	14600	174000	5	Nil	2.2	E.Coli Grown	No Growth	301	2.3	7.4	Neg	NAD	Y	Class II	Piperacillin plus tasobactam and Insulin	Peritoneal Dialysis	Nil	Survived

6	Arokiya dass	72	M	Y	Y	Y	Y	Y	24Yrs	N	Y	Y	17400	47000	15	Pos	4.6	Klebsiella Grown	Klebsiella Grown	474	6.2	10.6	Neg	NAD	Y	Class IIIB	Meropenem, Ceftriaxone and Insulin	Hemo Dialysis	CT guided drainage	Died
7	Kaveri	47	F	N	Y	Y	Y	Y	8Yrs	N	N	N	12300	162000	5	Nil	2.4	E.Coli Grown	No Growth	306	2.4	7.6	Neg	NAD	Y	Class II	Piperacillin plus tasobactam and Insulin	Peritoneal Dialysis	Nil	Survived
8	Saradhammal	72	F	N	Y	Y	Y	Y	25Yrs	N	N	N	18200	56000	15	Pos	4.2	Klebsiella Grown	Klebsiella Grown	448	5.9	10.6	Neg	NAD	Y	Class IIIB	Piperacillin plus tasobactam and Ceftriaxone and Insulin	Hemo Dialysis	Nil	Died
9	Murugammal	52	F	N	Y	Y	Y	Y	8Yrs	N	N	N	13600	154000	10	Nil	2.8	E.Coli Grown	No Growth	298	3.2	7.6	Neg	NAD	Y	Class I	Meropenem and Insulin	Peritoneal Dialysis	Nil	Survived
10	Rose	58	F	N	Y	Y	Y	Y	7Yrs	N	N	N	13800	182000	5	Nil	3.2	E.Coli Grown	No Growth	268	3.9	7.5	Neg	NAD	Y	Class I	Ceftriaxone and Insulin	Peritoneal Dialysis	Nil	Survived
11	Sidharthan	72	M	N	Y	Y	Y	Y	22Yrs	YBPH	N	YTurpdone	19200	54000	20	Pos	4.2	E.Coli Grown	E.Coli Grown	467	7.4	9.8	Neg	NAD	Y	Class IIIA	Meropenem and Insulin	Hemo Dialysis	Partial Nephrectomy of right kidney	Died

12	Parvathi	52	F	Y	Y	Y	Y	Y	9Yrs	N	N	N	14200	140000	10	Nil	2.8	E.Coli Grown	No Growth	257	3.6	7.8	Neg	NAD	Y	Class II	Piperacillin plus tasobactam and Ceftriaxone and Insulin	Peritoneal Dialysis	Nil	Survived
13	Kuttiyammal	72	F	Y	Y	Y	Y	Y	20Yrs	N	N	N	18200	70000	15	Pos	4.7	Klebsiella Grown	Klebsiella Grown	403	7.7	9.4	Neg	NAD	Y	Class IIIA	Meropenem, Vancomycin and Insulin	Hemo Dialysis	Nil	Died
14	Ammavasai	54	M	N	Y	Y	Y	Y	12Yrs	N	N	N	12200	156000	10	Nil	2.4	E.Coli Grown	No Growth	362	2.4	7.9	Neg	NAD	Y	Class II	Piperacillin plus tasobactam and Insulin	Peritoneal Dialysis	Nil	Survived
15	Kumari	52	F	Y	N	Y	Y	Y	11Yrs	N	N	N	12800	172000	5	Nil	2.8	E.Coli Grown	No Growth	322	2.6	7.2	Neg	NAD	Y	Class II	Meropenem and Insulin	Hemo Dialysis	Nil	Survived

16	Shanthi	57	F	Y	Y	Y	Y	Y	7Yrs	N	N	N	13300	210000	10	Nil	2.6	E.Coli Grown	No Growth	288	3.2	7.8	Neg	NAD	Y	Class II	Meropenem and Insulin	Peritoneal Dialysis	Nil	Survived
17	Abirami	58	F	Y	Y	Y	Y	Y	8Yrs	N	N	N	12600	115000	8	Nil	1.6	E.Coli Grown	No Growth	354	2.1	7.4	Neg	NAD	Y	Class I	Piperacillin plus tasobectam and Insulin	Peritoneal Dialysis	Nil	Survived
18	Rasathi	62	F	N	Y	Y	N	Y	22Yrs	N	N	N	19400	48000	15	Pos	4.1	E.Coli Grown	E.Coli Grown	472	8.6	9.8	Neg	NAD	Y	Class IIIB	Meropenem and Insulin	Hemo Dialysis	Nil	Died
19	Anjalai	73	F	Y	Y	Y	Y	Y	20Yrs	N	N	N	19200	52000	12	Pos	5.2	Klebsiella Grown	Klebsiella Grown	482	6.7	9.5	Neg	NAD	Y	Class IIIB	Piperacillin plus tasobactam and Ceftriaxone and Insulin	Hemo Dialysis	CT guided drainage	Died

20	Perumal	54	M	Y	Y	Y	Y	Y	8Yrs	YBPHTURPdone	N	YTurpdone	12800	160000	4	Nil	2.4	E.Coli Grown	No Growth	356	1.5	7.2	Neg	NAD	Y	Class I	Amikacin and Insulin	Peritoneal Dialysis	Nil	Survived
21	Arundathi	53	F	N	Y	Y	N	Y	4Yrs	N	Y	N	13500	130000	2	Nil	3.6	E.Coli Grown	No Growth	332	4.1	7.8	Neg	NAD	Y	Class II	Piperacillin plus tasobactam and Ceftriaxone and Insulin	Hemo Dialysis	Nephrectomy done	Survived
22	Arunagiri	49	M	N	Y	Y	Y	Y	7Yrs	YUrinary Stricture	N	N	14400	148000	7	Nil	2.6	E.Coli Grown	No Growth	307	32	7.2	Neg	NAD	Y	Class II	Meropenem and Insulin	Peritoneal Dialysis	Nil	Survived
23	Punniyakoti	56	F	Y	Y	Y	Y	Y	9Yrs	N	N	N	12600	196000	5	Nil	2.2	E.Coli Grown	No Growth	326	3.9	8.6	Neg	NAD	Y	Class I	Impeinen and Insulin	Peritoneal Dialysis	Nil	Survived

24	Poongodhai	74	F	Y	Y	Y	Y	Y	24Yrs	N	N	N	19600	62000	20	Pos	4.7	Klebsiella Grown	Klebsiella Grown	448	7.6	9.8	Neg	NAD	Y	Class IIIA	Piperacillin plus tasobactam and Ceftriaxone and Insulin	Hemo Dialysis	Nephrectomy done	Died
25	Muthammal	52	F	Y	Y	Y	Y	Y	8Yrs	N	N	N	12300	174000	10	Nil	2.8	E.Coli Grown	No Growth	347	4.2	7.4	Neg	NAD	Y	Class I	Meropenem and Insulin	Peritoneal Dialysis	Nil	Survived